THE MEDICAL JOURNAL OF AUSTRALIA

VOL. I.—23RD YEAR. SYDNEY, SATURDAY, JUNE 20, 1936. No. 25.

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THE MODERN OUTLOOK ON NUTRITION.

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DURING the past few years there has been a redistribution of the emphasis on the various aspects of the biological science of nutrition. During the past few months this process has reached a climax.

The purpose of this discussion is to trace the steps leading to the present position and to forecast the future developments, with their far-reaching economic effects.

The history of nutrition, as seen through the eyes of today, might be divided into six periods: (i) the period of the heat engine concept, (ii) the vitamin period, (iii) the biological period, (iv) the mineral period, (v) the period of balanced composition, (vi) the period of action. The first four periods might be termed the analytic phase; the last two are the synthetic phase. While all these periods overlap, they form a convenient scaffolding on which to trace the development and fruition of the modern outlook. The essence of the modern outlook on nutrition today is that there is no single measure of food value. A single foodstuff can be evaluated only in so far as it is a part of a combination that is qualitatively complete and quantitatively sufficient. Our existing knowledge of food composition is still incomplete; but at the same time it is sufficiently far advanced to indicate the serious shortcomings of the diets in common use.

The Period of the Heat Engine Concept.

The foundations of the knowledge of nutrition were laid down by the physicist, who studied the problem from the calorimetric and ergometric angles. The heat engine attitude was perhaps not to be wondered at, for it was simple and attractive; so simple, indeed, and so attractive was it that it

has taken the best part of a century for science to restrict its significance and to appreciate its fallacies. An unfortunate legacy from this period was the term "energy-producing" in relation to foods. To the physicist "energy" has a very real scientific meaning in relation to an engine: force moved through a distance. To others who have an outlook different from the physicist there is a danger that "energy" will be construed as the desire to do work. While animal heat production and muscular energy could be satisfactorily studied by calorimetric and ergometric means in the case of the human, mental effort was outside the scope of physical measurement.

The fallacy of adopting the calorie as the chief standard of food value is evident in the differing figures recommended to maintain efficiency by three different committees appointed in recent years by the Ministry of Health, the British Medical Association and the League of Nations.

The Vitamin Period.

When vitamins were discovered it was not realized that a foodstuff or vitamin preparation might seem to have a greater value when added to a markedly deficient ration than when added to a nearly perfect one. Especially is this the case under certain pathological conditions. The simile might be used of the addition of the fourth leg to a chair. The fourth leg completes the balance of the whole, but there is a danger that the fourth leg will receive all the credit for the stabilization of the equilibrium, which must be equally shared between all four.

The physicists maintained their heat engine analogy by likening the vitamins to the "vital spark" which activated the process. They mistakenly described them as "accessory food factors".

For many people the page of this period has not yet been turned over, as witness the prevalent use of concentrated vitamin preparations.

The Biological Period.

The appreciation of the differing biological values of protein was the first real blow to the pure heat-engine concept. An even greater blow was the biological value of combinations of proteins. In the feeding of pigs, for instance, the combination of maize and skimmed milk gives much better results than the use of either alone.

In this period it is convenient to include the recognition of the "protective foods". The mental processes involved in the coining and adoption of this name are interesting. These foods are milk, meat (including liver and kidney), eggs, green and leafy vegetables, potatoes, raw fruit and vegetables, and cod liver oil. On account of the biological balance of various factors present in their combination, they "protected" against the results of the inherent deficiencies of the dietaries in common use that were based on the more primitive and limited conception of the body as a heat engine. This was the most significant development in the whole history of nutrition, and actually a combination of these foods is the foundation of the balanced composition of the dietaries of today.

The Mineral Period.

The serious clinical results of iodine deficiency, the appreciation of the importance of the calcium-phosphorus ratio, and of the value of the balance of alkaline and acid ash foods in the diet directed attention to an aspect of the nutritional problem that was definitely novel. In the past, food manufacturing effort was directed to refining the "useless" from the "useful" elements in food. The drive for "purity" in foods certainly provided the calorie, but, to parody a legal formula, it gave us the calorie, the whole calorie and nothing but the calorie. Even today in the milling trade the mineral-rich constituents of the wheat berry are described as "offal".

Consideration of the mineral elements in the diet has opened up important problems of soil deficiencies, and Orr, of the Rowett Institute in Aberdeen, has directed attention to the limiting factors imposed by the varying mineral resources of the soil on agriculture, on animal nutrition, and, finally, on human nutrition.

By what may be described as a strange coincidence, the selection of those foods in the human dietary range of possibilities that are rich in mineral salts provides also a happy balance of other factors. Such foods include milk, meat, fish, cheese, eggs, potatoes, green and leafy vegetables, fruits and whole grain cereals.

The Period of Balanced Composition.

As a coping stone on the nutritional edifice as we see it today it is only natural that the best should have been selected from these preceding four periods, and from them a qualitative basis should have been deduced. What still remains to be completed is the quantitative relationship which these factors should bear one to the other; and here already definite guidance is available. As regards total daily quantity of food, in the healthy subject and with a qualitatively and proportionately balanced diet, appetite adjusts food intake to biological requirements. It is chiefly when the balance is upset by the excessive consumption of an unbalanced foodstuff like sugar that the appetite guide becomes fallacious. The League of Nations Commission on the Physiological Bases of Nutrition[1] states: "The excessive use of sugar is to be condemned."

The basis of the modern outlook is "The Law of Maximum Normal Nutritive Value":[2]

> An individual foodstuff expresses its normal and most characteristic nutritive value, for a given kind of animal, under specified conditions governing nutritive requirement, only as it is a part of a ration which is qualitatively complete and quantitatively sufficient, for the conditions existing—except as it may express the same value by virtue of the capacity of the animal temporarily to protect itself from food nutrient deficiency by drafts upon the nutritive reserves of its own body, or as it may express even higher apparent value, under certain patho-

logical conditions, or during undernutrition, by virtue of the protective or body-sparing capacities of nutrients.

This new conception of balance in nutrition is evident in Sir John Orr's report on the adequacy of the food habits of the British people. On a basis of calorie value alone, only 12% of the population have an inadequate intake, but when the intake of the population is compared with Stiebeling's and Sansum's standards of calcium, phosphorus, iron, vitamin *A* and vitamin *C*, it can be shown by the accompanying graph that 50% of the British people fall below the standard of adequacy. The two accompanying charts make this position clear.

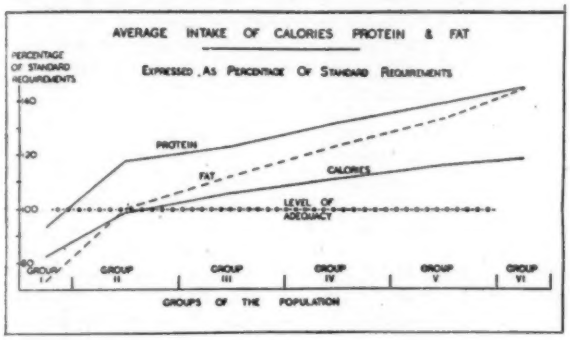

CHART I.

These are taken from "Food, Health and Income."[3] The principle of balance is also evident in Table I, showing quantities of nutrients required for individuals per day.

With so many factors entering into the composition of a standard balanced dietary, it is best to simplify the situation by expressing the balanced composition qualitatively in terms of actual foodstuffs, and quantitatively in terms of proportionate amounts. These amounts also serve as a guide to the optimum requirements of nutrition (in the highest sense of the word). In view of the public health importance of nutrition, it is best that the modern outlook on nutrition be expressed in simple

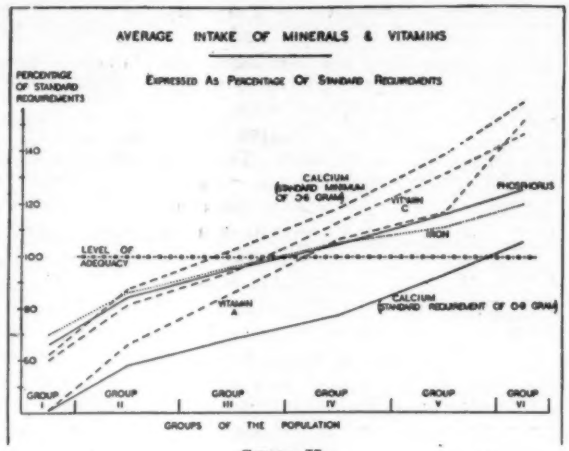

CHART II.

language that can be readily understood. From the standpoint of the public also there is a danger that an attempt to explain the complex factors entering into adequate nutrition will bring about too great a preoccupation with diet. After all, food is to eat and to enjoy. Too much consideration of the underlying principles of nutrition tends to reduce the art of dining to the level of a visit to the medicine cabinet.

The following simplified statement has been prepared from the report of the League of Nations Commission on the Physiological Bases of Nutrition and the League of Nations publication, "Nutrition and Public Health", by Burnet and Akroyd. It has been adapted from these in accordance with the special nutritional environment which Australia affords.

Foodstuffs are of two kinds: foundation foods and supplementary foods. There are five foundation foods. They are milk, meat, the dairy products, fruit and vegetables. While a simplified diet can be devised from these foods so as to be completely satisfactory, variety in diet tends to safety.

TABLE I.

Quantities of Nutrients Required for Individuals per Day.

(Stiebeling.)

Individuals by Age, Sex, and Activity Groups.	Dietary Allowance in:						
	Energy Value.	Protein.	Calcium.	Phosphorus.	Iron.	Vitamin *A*.	Vitamin *C*.
	Calories.	Grammes.	Grammes.	Grammes.	Grammes.	Sherman Units.	Sherman Units.
Child under 4 years	1,200	45	1·00	1·00	0·006 to 0·009	3,000	75
Boy 4 to 6 years; girl 4 to 7 years	1,500	55	1·00	1·00	0·008 to 0·011	3,000	80
Boy 7 to 8 years, girl 8 to 10 years	2,100	65	1·00	1·00	0·011 to 0·015	3,500	85
Boy 9 to 10 years, girl 11 to 13 years	2,400	75	1·00	1·20	0·012 to 0·015	3,500	90
Moderately active woman, boy 11 to 12 years, girl over 13 years	2,500	75	1·00	1·20	0·013 to 0·015	4,000	95
Very active woman, active boy 13 to 15 years	3,000	75	0·88	1·32	0·015	4,000	100
Active boy over 15 years	3,000 to 4,000	75	0·88	1·32	0·015	4,000	100
Moderately active man	3,000	67	0·68	1·32	0·015	4,000	100
Very active man	4,500	67	0·68	1·32	0·015	4,000	100
Average per head of the population	2,810	68	0·9	1·23	0·013 to 0·014	3,800	95

TABLE II.

Food.	Ozs.	Grms.	Calcium (Grms.)	Phosphorus (Grms.)	Iron. (Milligrms.)	Iodine. (Milligrms.)	Vitamins (International Units).					Protein. (Grms.)	Fat. (Grms.)	Carbohydrate. (Grms.)	Calories.
							A.	B_1.	B_2.	*C.*	*D.*				
A. *Foundation Foods:*															
Milk (1½ pints) ..	30	900	1·08	0·81	2·16	0·018–0·045	2,700	45–135	Rich.	Poor.	Poor.	30·0	30·0	45·0	570
Meat (including fish, liver, kidney or poultry) ..	4	120	—	0·30	3·0/2	..	Poor.	Poor.	Rich.	Poor.	None.	28·0	12·0	0	212
Eggs (1)	2	60	—	0·12	1·7	..	1,560	16	Rich.	None.	48	12·0	12·0	0	156
Cheese	2	60	0·6	0·4	0·8	..	1,600	Poor.	Good.	Poor.	Poor.	16·0	22·0	0	262
Green and leafy vegetables ..	3	90	0·45	—	0·9	..	90	23 (raw).	Good.	270 (raw).	None.	1·5	0	3·0	18
Potatoes	8	240	—	0·2	3·0	..	Poor.	48	Good.	150	None.	8·0	0	48·0	224
Raw fruit and vegetables ..	8	240	—	0·2	3·0	..	Poor.	48	Good.	500	None.	4·0	0	16·0	80
Butter	2	60	—	0·2	3·0	..	Good.	..	Good.	..	None	0	50·0	0	450
Totals	59	1,770	2·13	2·23	13·06	0·25	6,260	205	Adequate.	920	48	99·5	126	112	1,972
B. *Supplementary Foods:*															
Cereals as needed—															
Whole grain ..	8	250	—	0·9	9·0	..	..	270	..	..	None.	24·0	0	144·0	1,000
or															or
Lightly milled ..	8	250	—	0·4	6·0	..	..	140	..	..	None.	24·0	0	144·0	1,000
															2,972

As a general average for all ages from two years old the daily quantities of these foundation foods should be: milk, one and a half pints; meat, including fish, liver, kidney or poultry, four ounces; egg, one; cheese, two ounces; green and leafy vegetables, three ounces; potatoes, eight ounces; raw fruit and vegetables, eight ounces; butter, two ounces. (See Table II.)

As regards the supplementary foods, of which the chief is the cereal, white flour in the process of milling is deprived of important nutritive elements. Its use should be decreased and partial substitution by lightly milled cereals and especially by potatoes is recommended. Children, pregnant and nursing women should consume wholemeal bread. The greater the quantity of cereal in the diet, the more important it becomes that the more nutritive parts of the grain should be consumed. The consumption of an excess amount of sugar is to be condemned. Skimmed and separated milk has a high nutritive value, and is specially valuable to those who are anxious not to be overweight. Caution should be exercised in the use of concentrated vitamin preparations, as they do not, except in special cases under medical direction, make up for the deficiencies of the usual diets in use at present.

The foods indicated will provide all the vitamin and mineral constituents necessary, and will, moreover, provide the balance that is so fundamental in adequate nutrition.

It is important to appreciate the fact that a diet which may keep an adult living an unexacting life in fair health may fail to meet the requirements of a growing child and be inadequate for times of stress (hard manual work, pregnancy, infectious disease *et cetera*).

This is not a diet. It is the father of all diets. It is like a suit length which is of a standard width and length and of more or less standard material, but which must be cut and fitted to the individual's peculiar requirements. The principles of balance and the proportion of the foods are more important than the details.

This dietary schedule will need to be varied in accordance with age, consideration being given to whether the individual is living in a warm or cold climate or season, and whether he is performing light, moderate or heavy work. In regard to age, it must be appreciated that the growing child requires a more carefully balanced diet and a greater intake than his body weight in proportion to the adult would indicate. The diet assumes an even greater importance when the individual is undergoing the nutritional exigencies of pregnancy and lactation, but the general principles remain the same throughout.

The Period of Action.

The reduction of dietary essentials to a form that is simple and yet one which embraces all the schools of thought of the previous four periods, paves the way for a definite plan of action which cannot fail to have a profound effect on public health and economics. In Australia the environment is particularly suitable for the widespread application of these nutritional principles. Australia is a primary producing country and is largely free of the nutritional disadvantages of those countries which are colder, which are less sunny, and which have a greater population density. Especially in the more populous fringes of our island continent we have ample supplies of the foundation foodstuffs available at a price that is within the reach of a greater proportion of our population than is the case with European and American countries. We have the opportunity to become, nationally, the prototypes of nutritional excellence. Actually, a review of the food situation in Australia shows a staggering dis-

agreement between available foodstuffs and their utilization by the population. The following figures are obtained from Sir John Orr's book and from Commonwealth official statistics. The figures in each case for the year 1934 per head of population are shown in Table III.

TABLE III.

Substance.	Britain.	Australia.
Meat	143 lbs.	186 lbs.
White flour	197 lbs.	202 lbs.
Sugar	94 lbs.	106 lbs.
Cheese	10 lbs.	4½ lbs.
Butter and butter substitutes	33 lbs.	32 lbs.
Potatoes	210 lbs.	122 lbs.
Milk	80 quarts.	72 quarts.
Fruit	9 pence per person per week.	3½ pence per person per week.
Vegetables (excluding potatoes)	4½ pence per person per week.	1½ pence per person per week.

It will be noted that Australians consume less milk, half the cheese, half the potatoes, and less butter and butter substitutes combined than people in England. Even allowing for the difference in cost of fruit and vegetables as between Great Britain and Australia, the low expenditure on fruit and vegetables is a national and nutritional disgrace. Our higher consumption of meat requires to be balanced by an increased consumption of milk, fruit, vegetables and the dairy products.

The Australian basic diet is meat, potatoes, scanty overcooked vegetables, sweet cake, white bread and jam, and tea with sugar. There is nothing revolutionary in the statement that this diet is grossly unbalanced and markedly deficient in essential constituents. Sir Robert McCarrison, at the Pasteur Institute in India, using thousands of rats, showed years ago that rats fed on such a diet suffered from tooth decay, catarrhal diseases, respiratory disease, gastric ulcer and cancer, while rats fed on a balanced diet of meat, vegetables, milk and wholemeal bread were free from such troubles and remained healthy.

Sir John Orr has shown that before nutritional saturation is reached high percentage increases of milk, butter, eggs, fruit and vegetables are possible. These possibilities have been provisionally estimated for Australia with the following result: milk, 90%; butter, 36%; cheese, 50%; fruit, 124%; potatoes, 50%; vegetables (excluding potatoes), 150%; meat, saturated consumption; wholemeal flour, 100%.

From the nutritional standpoint also decreases are advisable in the remaining foodstuffs. Particularly in tropical and subtropical climates is a marked reduction of the refined carbohydrates essential.

While Australia keeps on with its present unsatisfactory diet, it will never have a first rate population. In this regard it is interesting to quote Dr. J. V. Duhig in his presidential address to the Section of Medical Science and National Health at the last meeting (Melbourne, 1935) of the Australian and New Zealand Association for the Advancement of Science:

> The improper use which we have been making of our food supply is little short of staggering. The basis of a national dietary should be milk, meat, butter, eggs, fresh fruit and fresh vegetables. The application of the principle that disease can be caused by the absence of necessary factors from a dietary seems to offer promise of bringing actual disease to a fraction of its present extent by the use of rational dietaries.

The established standard of adequate nutrition gives a new significance to the term malnutrition. The indications are that much of our medical and surgical knowledge will have to be reconsidered in the light of this new outlook. Tooth decay, with all its "septic foci" ramifications throughout the body, catarrh, sinus disease, alimentary ulcerations, and even cancer of the stomach are just a few of the common disabling diseases which will have to be viewed afresh from this new angle. Tooth decay, indeed, can now be regarded as an index of malnutrition. Australia, weighed in the balance of this new standard of balanced nutrition, has been found wanting. There is scientific justification for the marked extension of the dairying, fruit-growing and market-gardening industries; there is a similar justification for the reduction of the flour milling, sugar growing, biscuit manufacturing and confectionery industries. As Dr. S. F. McDonald said at the meeting of the Queensland Branch of the British Medical Association on May 1:

> There are three Australian industries which sacrifice the children's teeth as the price of their success: the sugar industry of Queensland, the biscuit industry of New South Wales, and the confectionery industry of Victoria.

Mr. S. M. Bruce, speaking to the League of Nations last September, sums up the new outlook with his phrase: "The marriage of public health and agriculture." In the words of the leading article in *The Lancet*:[4]

> The first step is to ensure that the marriage now arranged between public health and agriculture shall shortly take place.

Acknowledgements.

In the preparation of this paper grateful acknowledgement is made of the valued assistance of Professor E. J. Goddard, Professor of Biology and Dean of the Faculty of Agriculture, University of Queensland; Professor J. K. Murray, Professor of Agriculture; Professor F. Helmore, Dean of the Faculty of Dentistry; Dr. A. Earnshaw; Dr. Mathieson; Mr. H. J. G. Hines, Lecturer in Bio-Chemistry; and my assistant, Mr. M. Powell, B.Ag.Sc.

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(1) The Report of the League of Nations Commission on the Physiological Bases of Nutrition, Geneva, December 9, 1935. Published in *The Lancet*, December 21, 1935.

(2) E. B. Forbes: "The Law of Maximum Normal Nutritive Value", *Science*, Volume LXXVII, Number 1995, March 24, 1933, page 306.

(3) J. B. Orr: "Food, Health and Income", 1936.

(4) *The Lancet*, Leading Article, January 4, 1936, page 31.

THE TREATMENT OF CIRCULATORY FAILURE.

By H. W. WUNDERLY,
Adelaide.

WHILE it is true that the whole cardio-vascular system is one unit and must be considered as such, it is convenient to divide it into central and peripheral parts. This is especially important when considering the treatment of circulatory failure, for, although the circulation fails as one system, it is usual for one part to fail before the other. Clinically, it is essential to know whether one is faced with a condition of central or heart failure or of peripheral failure.

Warfield,[1] in a very thoughtful paper, has drawn attention to the work of Wollheim.[2] Wollheim divides cardiac decompensation into two groups, which he calls *plus* and *minus* decompensation. The chronic *plus* decompensation cases are those in which breakdown is the result of excessive strain in hypertonia, aortic insufficiency, most cases of mitral stenosis, especially when combined with insufficiency, and large left ventricle. This is the more common form and is characterized by dyspnœa, orthopnœa, œdema, distension of the veins in the neck and cyanosis. These patients are more comfortable sitting up with legs hanging down, as the blood volume is reduced from 400 to 1,000 cubic centimetres by filling of the subpapillary skin plexus of the legs.

The chronic *minus* decompensation cases are seen in primary disturbances in oxygen exchange in the lungs in pulmonary stenosis, pulmonary sclerosis, not infrequently in mitral stenosis, in emphysema and in thyreotoxicosis. In the acute form they are seen in pneumonia, lobar pneumonia and bronchopneumonia of influenza, toxic infectious diseases, and in circulatory shock. The most striking characteristic of the chronic *minus* cases is the lack of dyspnœa at rest. These patients are comfortable lying flat in bed. There is no distension of the neck veins. Cyanosis is found in patches in the skin, whereas in the chronic *plus* cases it is most marked in the lips and acral parts. The blood volume may be as low as 40 to 50 cubic centimetres per kilogram of body weight. These cases are considered to be due to vascular insufficiency with blood held back in the depôts, in contradistinction to cardiac insufficiency, in which more blood is actually in circulation.

The treatment of central or cardiac failure is along the usual lines of digitalis in adequate dosage, strophanthin, diuretics and venesection. When cyanosis is present, great benefit is derived from the use of the oxygen tent or oxygen administered through a nasal catheter. Many physiologists deny the value of this latter method, but when the catheter is pushed into the naso-pharynx and the oxygen is administered at the rate of two litres a minute the effect is often dramatic. Besides oxygen, the heart muscle needs a liberal supply of glucose and insulin, and many failing hearts are improved by glucose administered intravenously with small doses of insulin. The administration of oxygen, insulin and glucose diminishes the amount of lactic acid in the blood in these cases of heart failure.

The circulatory failure of the infectious fevers, of circulatory shock and of severe hæmorrhage requires a totally different treatment, for in these conditions the failure is peripheral and not central. It is not the heart which fails primarily. It is known that the heart receives its stimulus to contract from an adequate diastolic filling and that there must be a certain head of pressure in the coronary circulation for the heart to do its work efficiently. But in the circulatory failure of the infectious fevers there has been a loss of fluid into the tissues and so the blood volume has been diminished and there has been a loss of chlorides from the blood. The blood has become concentrated and the venous pressure diminished.

Owing to the diminished blood volume and the low venous pressure the ventricle does not become properly filled in diastole and the heart beats faster to keep up the minute volume of blood in circulation. A point is reached when the diastolic pressure will not provide an adequate circulation in the coronary vessels; lactic acid accumulates in the heart muscle, which then dilates and fails.

While on the subject of intravenous therapy, it might not be out of place to say something about the preparation of the solutions used, and of the apparatus, also about the various conditions in which this form of therapy is useful. Banks[3] states that "the hyperpyrexia associated with chill and fever following the intravenous administration of fluids has been a disturbing factor in this method of treatment". As far back as 1923, Seibert[4] showed that distilled water was likely to become infected on standing. She pointed out that if the water was sterilized within twenty-four hours of its being distilled it was not pyrogenic and did not become so if its sterility was maintained. She found[5] that the substance causing the intravenous reaction is specific for that type of reaction and that it is derived from the bacterial bodies and products of growth of *Pseudomonas scissa* and *Pseudomonas ureæ*. The pyrogen substance is water-soluble and is rapidly eliminated by frequent washings with pyrogen-free water. Elser and Stillman[6] showed that the use of triply distilled water in the preparation of intravenous solutions is not necessary. One distillation is sufficient if the still is fitted with a spray trap. Lee Rademaker[7] calls attention to the preparation of the rubber tubing in order to avoid reactions after intravenous infusions. He suggests filling the tubing with a 10% solution of sodium hydroxide for twelve to twenty-four hours, washing it in running water for two hours, and boiling in distilled water for half an hour just before using. In large hospitals it is possible to have the solutions and apparatus prepared in a special solution department, but this is not possible in smaller establishments. The various solutions are now put up in specially sealed

flasks all ready for use, and are supplied with sterilized sets of rubber tubing, needles, drip control, clamp and even thermometers to measure the temperature of the fluid just before it enters the vein. These flasks would be invaluable in our private and country hospitals.

In cases of hæmorrhage, not only is the volume of blood diminished, but there is a great diminution in the amount of oxygen carrying hæmoglobin. The most satisfactory method of increasing blood volume is by giving fluids intravenously. The ideal fluid is blood or blood plasma. But a large supply of blood suitable for use in a particular case is not always to hand. Suitable solutions of dextrose or dextrose and saline solution can be available at any hour of the day or night. The disadvantage of these two solutions is that they do not remain very long in the circulation. What is wanted is a fluid which contains colloids. Not only does a blood transfusion increase the blood volume, but it supplies the necessary ions of calcium, sodium and potassium and also oxygen-carrying red blood corpuscles. If anæmia is not present, a very useful colloidal solution is a 6% acacia solution.

It is only during the last few years that it has been realized that fluids should be given in much larger quantities than had been done in the past.[8] Three or four litres can be given in twenty-four hours. In order to avoid "speed shock" it is best to introduce the fluid at the rate of two or three cubic centimetres per minute, that is, about forty drops a minute. The temperature of the injected fluid is not very important. If the fluid is given at the above rate its temperature matters very little and many hospitals use it at room temperature. The ideal temperature would be somewhere about 44·4° C. (112° F.). If the patient is feverish it could be given at a temperature of about 25·5° C. (80° F.), but if the object is to combat shock a warmer solution must be used. Also in shock it is permissible to inject the first few hundred cubic centimetres at the rate of ten to twenty cubic centimetres per minute, a careful watch being kept on the pulse and the blood pressure.

When excessive sweating, vomiting, diarrhœa or exudation (for example, in burns) accompanies the loss of fluid from the circulation it is best to use a 5% dextrose in saline solution to replace the salt loss. It is unnecessary to use a salt solution for a patient who is only thirsty.

The intravenous route is especially useful when vomiting or diarrhœa render normal intake by the mouth useless, when gastric or intestinal hæmorrhage calls for complete rest to the parts, in the hypoglycæmia of "insulin shock" and in thyreotoxicosis. In the last condition iodine can be added to the dextrose solution.

Osmotherapy, by which is meant the administration of hypertonic solutions, is useful in the treatment of pulmonary œdema of myocardial origin and in the treatment of increased intracranial pressure when slow pulse, low blood pressure and stertorous breathing are present. For these conditions a 25% solution of dextrose is most useful. Only in those states in which there is a sodium chloride deficiency should saline solution be used. One litre of physiological salt solution contains 8·5 grammes of sodium chloride, and 2·0 grammes are sufficient for an average person. So stop the use of saline solution when sodium chloride reappears in the urine. The use of salt is indicated in the treatment of hypochloridæmia of intestinal obstruction, profuse perspiration, heat cramps and when profuse exudation has taken place, as in severe burns. Normal saline solution is bad for patients who are thirsty. Apart from the cases in which there is a sodium chloride deficiency, dextrose solution should be used.

To return to the treatment of circulatory failure of the peripheral type. It was stated that the best method of increasing the blood volume was by fluid given intravenously. There are some drugs which are said to increase blood volume. These are strychnine, camphor, cardiozal, caffein, adrenaline and pituitrin. Warfield[1] gives strychnine in heroic doses and advises 4·3 to 6·5 milligrammes (one-fifteenth to one-tenth of a grain) of strychnine sulphate every hour or two for an adult. Bandaging the limbs and raising the foot of the bed both help in the treatment of these cases. There does not seem any conclusive evidence that digitalis acts as a heart stimulant, and, in that it diminishes blood volume, it is contraindicated in the treatment of these cases.

Summary.

1. Circulatory failure has been divided into (*a*) central or cardiac, and (*b*) peripheral.

2. Central failure is usually accompanied by increased blood volume, peripheral failure by diminished blood volume.

3. Following the classification of Wollheim, cardiac failure was divided into *plus* decompensation, which is characterized by dyspnœa, orthopnœa, cyanosis of lips and acral parts, and increased venous pressure; and *minus* decompensation, in which there is low blood volume, no dyspnœa on lying down, patchy skin cyanosis and low venous pressure.

4. The acute cases of *minus* decompensation correspond to circulatory failure in the severe infections. *Minus* decompensation is a peripheral circulatory failure, not a failure of the heart itself. In it four conditions are found: (*a*) decreased blood volume and insufficient venous return flow, (*b*) concentration of the blood, (*c*) decreased blood chlorides, and (*d*) low venous pressure.

5. The treatment of peripheral circulatory failure is the treatment of these four conditions.

6. The most satisfactory method of increasing blood volume is by introducing fluids intravenously. These should be administered at the rate of two or three cubic centimetres per minute, and large quantities are usually necessary. Blood is the best fluid; 6% acacia solution is useful if the hæmo-

globin is not below 25%; up to three or four litres a day should be given.

7. The cause of the hyperpyrexial reaction was stated and methods of preparation of solutions and apparatus were given.

8. The drugs that are recommended are those that increase blood volume.

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SOME SURGICAL ASPECTS OF THE GOITRE PROBLEM.[1]

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BEFORE discussing the selection of cases for operation and their management there are certain conditions to which I wish to draw attention in the prevention of serious complications of thyreoid disease. If these were clearly recognized by the profession at large there would be less thyreotoxicosis in middle life, that is, at the age period when the risks of the necessary treatment are greatest.

Adenoma of the Thyreoid.

The thyreoid provides us with an example of how single or multiple adenomata can occur in an endocrine gland. The growth of an adenoma mimics the parent gland very closely, and, being encapsuled, it remains just as separate from the rest of the thyreoid substance as if it were growing in another region; like the breast adenoma, however, it is likely to share in the physiological enlargements of the parent gland. At the same time we must always bear in mind that the adenoma is not under the usual vascular or nervous control of the gland proper, and that its structure may remain in the undeveloped fœtal state. I know of no other type of growth in which the patient is told so often by the family doctor, and even by the specialist physician, that the best thing to do is to forget about it, as it will never do any harm. In my experience thyreoid adenoma has more chance of harming the individual than any other type of "simple" growth. Certainly there is not the same tendency for such a tumour to undergo carcinomatous change, as, for instance, in the parotid or breast, but the tumour may either change its own secretory activity or by its presence induce such degenerate and fibrotic changes in the gland proper as will induce a definite alteration in the gland's secretory activity.

Patients may have tumours of the thyreoid in evidence for five to twenty years or more and experience no trouble, but toxic symptoms may then supervene, and often the onset is most insidious. I always hold that the lucky folk are those who notice their symptoms and who seek surgical relief before the menopause occurs; their toxæmia has become evident, they come earlier for treatment and are eminently curable. The unlucky folk are those who have had a mild toxæmia going on for many years and whose mild toxæmic symptoms are put down to a "nervous temperament". After the menopause these folk are prone to develop an acute cardiac breakdown resulting from chronic myocardial degeneration, and, unless they are referred very promptly for surgical treatment, they may never be restored to anything approaching normal.

It has been suggested that in cases such as these the thyreoid has become adjusted over a number of years to a low iodine intake. When the increase of hormones usually provided at the menstrual period is no longer required, the system generally is overloaded, the thyreoid is overcharged for its needs, and toxic symptoms result. One cannot but feel that in these cases of chronic thyreotoxicosis the toxin has a special affinity for heart muscle and induces definite myocardial degeneration.

In the past few years there seems to be a decided increase in the number of old goitres becoming toxic, and it is of interest to note that this coincides with the increased availability of iodine preparations to the general public. It is well known that these patients with toxic adenoma of the thyreoid not only fail to respond to iodine medication as do those with physiological enlargement or of true Graves's disease, but that iodine is actually harmful to them. Iodine in various forms has become a fashionable therapeutic agent for many ailments, particularly those to which the middle-aged are subject, such as rheumatic conditions, chronic pulmonary diseases, increased blood pressure *et cetera*; and the public is buying without prescription increasingly large quantities of medicine containing iodine. Among the broadcast virtues of these preparations is that of curing goitre, so that sufferers from thyreoid troubles frequently dose themselves before consulting a medical practitioner.

It seems that the young, or at least the healthy, thyreoid gland is able to accommodate itself to an excessive supply of iodine and that it rejects what is not wanted; but practitioners should exercise caution and discrimination in giving iodine to any patient over twenty-five years of age with any thyreoid enlargement, particularly if an adenoma is present in the gland. Plummer states that "the administration of iodine for a few days or a few

[1] Read at a meeting of the Central Northern Medical Association, New South Wales, on April 17, 1936.

weeks to patients over thirty years of age having adenomatous goitre often initiates hyperthyreoidism, which, once started, continues for months or years".

Apart from the question of malignant disease and thyreotoxicosis in relation to adenoma of the thyreoid, we also have disabilities due to the mechanical effect of the tumour. Pressure symptoms are complained of frequently, but most are of no serious moment. These tumours generally enlarge slowly, so that considerable dislocation of the trachea and larynx may result and at the same time cause no symptoms. But these large tumours are prone to hæmorrhage, which causes a rapid increase in size, leading at times to serious symptoms. In addition, if the pressure is considerable and extends over a long period, as in a retrosternal tumour, erosion or absorption of the cartilaginous rings of the trachea may occur; if this is of any great extent, it may permit of a "flapping" of the trachea when the tumour is removed.

Mention should be made of a peculiar feature sometimes met with in connexion with adenoma of the thyreoid, and that is the possibility of its developing metastatic growths with a predilection for the expanded ends of long bones. Clinically there can be little doubt that this condition is malignant, for, even though the thyreoid tumour may not enlarge appreciably, yet the behaviour of the metastasis is that of a malignant growth.

Ewing makes the following remark concerning these tumours: "Most metastasizing simple [*sic*] adenomas exhibit areas of adeno-carcinoma in some part of the tumour", which, to my way of thinking, is rather contradictory.

The first case of this type which I saw was in a woman of some sixty years of age who had had a large tumour of the thyreoid for forty years. She had had pain about the right knee joint for about eighteen months and was treated for rheumatism, but swelling of the lower end of the femur led to further investigation. X ray examination revealed an endosteal tumour producing great rarefaction, and biopsy proved the tumour to consist of tissue indistinguishable from thyreoid tissue and containing some colloid. She refused to have her old goitre touched and we ultimately lost sight of her.

Having drawn attention to certain happenings in the course of these "simple" or "benign" tumours of the thyreoid gland, one may briefly sum up as follows:

1. Clinicians should carefully avoid the prefix of "simple" or "benign" with regard to tumours of this gland, even if they are thought to be non-malignant.

2. Adenomata or their variations, for example, fibro-adenomata, cysto-adenomata or mixed adenomata, are potential foci upon which carcinomatous or other malignant metamorphosis may occur.

3. Apart from this question, such tumours may cause mechanical disabilities both as regards the comfort and appearance of the patient and in the functional activity of the gland.

4. It is our duty to our patients to warn them of the possible danger of leaving such tumours, and to advise them, if they are young, to have the tumour removed before they reach the danger age of twenty-five years; if they are beyond this age when the tumour develops, we should advise them to submit to operation without undue delay.

5. With all due deference I submit to our morbid anatomists that they too should discard the terms "simple" or "benign" in describing such tumours. If they wish to imply that there is no malignant change in the growth, they should make the statement in unmistakable terms or should use the prefix non-malignant.

Hyperthyreoidism.

In between the variations of the normal physiological reaction of the thyreoid gland and the abnormal and pathological reaction found in established thyreotoxic states, one meets with definite cases of hypersecretion, that is, with cases in which there is an excessive amount of normal secretion provided by the gland for unusually long periods. If you look for this condition you will find it to be common in many young or youngish women prior to menstruation as well as in both sexes at times of emotional unrest or distress.

Hypersecretion has probably been experienced by us all; it increases the speed of living, all our tissues are keyed up to give of their best, so that we are filled with a sense of well-being and wakefulness, the flexibility and elasticity of our mental and physical processes are stimulated, and we feel, think, and act clearly and quickly. Fortunately, such a demand for thyreoid hypersecretion is made only occasionally. Apparently the adrenals supply the preliminary energizing factor for any sudden critical call on the individual until the more slowly acting but much longer lasting thyreoid secretion can come into play. But with the thyreoid, as with all other tissues and organs, constant and excessive demands may lead to degenerative changes in the secreting cells and later to similar changes in the minute anatomy of the gland, resulting in dysthyreoidism or thyreotoxicosis. But during these periods of over-stimulation we must not lose sight of the fact that all bodily tissues share in the flogging, and breakdown in regions other than the thyreoid gland may occur, as instanced clinically by auricular fibrillation, glycosuria, alimentary crises, acute mania *et cetera*.

These cases of hyperthyreoidism are frequently associated with some degree of sympathetic imbalance, are eminently curable by suitable medical treatment, and should not be confused with or classed among the thyreotoxic states.

Thyreotoxicosis.

We shall now turn our attention to a consideration of thyreotoxicosis.

Ætiology.

Thyreotoxicosis has been very widely discussed and many theories as to its ætiology have been advanced; but, as with many other ailments, there do not seem to be any definite causative factors.

Dunhill divides the factors into three sections: (i) the stimuli, (ii) the thyreoid gland, and (iii) other body tissues. He states that each of these factors is capable of many variations, and different reactions are seen in different individuals, according to the variations and degree of predominance of the several factors. Thus he accounts for the multiplicity of clinical manifestations in what he believes is basically a single pathological entity.

It has been said that the sex epochs play an important part in thyreotoxicosis; but the only one I am sure of is the menopause, and I believe that a close analysis of the symptoms usually complained of as accompanying "change of life" will show many of them to be due to thyreoid imbalance. If sex epochs are an important factor, why do we not see many more cases of thyreotoxicosis at the age of puberty? Also, what is the sex epoch which influences the male, since the disease is much more common in males than is generally supposed?

The question of focal sepsis has been raised, but those of wide experience seem to agree that, if it is a factor in thyreotoxicosis at all, it is a secondary and not a primary one, its tendency being to aggravate a preexisting condition and not to initiate the disease. It may be placed in the same category as any prolonged or exhausting illness which makes a long-continued demand on thyreoid activity. If focal sepsis is present, it is better for the patient to deal with the thyreotoxic condition first and then to deal with any focal sepsis, not *vice versa*, as is so often advised.

There is no doubt that psychical trauma, especially when associated with physical insult, is a stimulus to the onset of the disease, but chiefly when the thyreoid tissue is in a state of imbalance or exhaustion.

It seems to me that the chief factor in ætiology is the condition of the gland itself, especially if it is the seat of multiple adenomata. In other cases a history can often be elicited of thyreoid enlargement in early childhood, which has apparently subsided about puberty or before the age of twenty years. Before leaving this question of ætiology I must quote Dunhill, who states:

> I believe that the stimuli which bring about the disease are situated without the gland and that these stimuli affect the gland, causing such change in its structure and function that its secretion is altered, and consequently other organs are intoxicated by its altered secretion.

Diagnosis.

Diagnosis is easy in the greater proportion of established cases; we are learning, however, to recognize not only the earlier manifestations, but many of the variations of the thyreotoxic state. The early signs and symptoms are chiefly those of over-stimulation of the sympathetic nervous system, and I would draw your attention particularly to a state of general excitability and nervous instability, evidenced by irritability, outbursts of temper, flushings, rapid breathing, fluctuations in weight, attacks of palpitation, periods of physical exhaustion, sweatings *et cetera*. Such patients are frequently referred to by their relatives and friends as having always been "highly strung", or are even classed by their medical advisers as being neurasthenic; but a careful history and investigation of the symptoms should lead one at least to suspect the true state of affairs. For some reason or other I have found the condition in its earlier stages overlooked more often in males than in females; but if doubt exists, it is now easy to obtain confirmation by an estimation of the basal metabolic rate. But do not send the patient for a test during a period of remission, which is a feature of the early stages, nor after a course of sedatives and iodine, or you may be misled by a reading within normal limits.

Prophylaxis.

Believing, as I do, that the main factor in ætiology is the state of the thyreoid gland, I would urge the importance of early removal of all adenomata or cysto-adenomata found, irrespective of the patient's age, as being the best means of preventing many of the thyreotoxic conditions met with about the middle-age period.

To all patients showing signs of hyperthyreoidism the condition and its possible dangers must be carefully explained, and they must be content to live quiet and even sheltered lives. In or just after periods of excitement or emotional stress, and prior to the menses, small doses of iodine for one to three days may be advisable, but many patients require bromide or other sedatives as well. By a careful regulation of their lives it is possible to carry these patients through the sthenic years of life until they reach the years of discretion which seem to come with recession of thyreoid activity. Be especially careful of these people during and just after the menopause, as they are then particularly liable to an acute thyreotoxic condition, in which auricular fibrillation may be the dominant factor.

Treatment.

Patients with true hyperthyreoidism respond well to rest, sedatives and freedom from excitement, worry and noxious stimuli, while small doses of iodine (for example, 0·06 to 0·18 cubic centimetre or one to three minims of Lugol's solution daily) may be given for periods of one to seven days. In view of the uncertainty existing in the minds of many practitioners regarding the use of iodine in goitre cases, I would ask you to pay special attention to my remarks on the subject. In the established thyreotoxic states in which operation is indicated, do not give iodine until the patient is in hospital and undergoing pre-operative treatment. In doubtful cases, iodine therapy is admissible only for very limited periods, not exceeding three weeks, and in minimal doses. Experience has taught me that the smaller the dose required to bring the patient to a stable level, the better the result for that individual. For the sake of obtaining a uniform standard I employ only Lugol's solution, and even in the worst cases seldom find it necessary

to give more than three drops three times daily. The main points to watch in giving iodine are: (i) the slowing of the pulse rate, (ii) the lessening of symptoms, (iii) the hardening of the thyreoid. If these occur you can be sure that there will be a coincident drop in the basal metabolic rate.

Once a toxic condition of the thyreoid has become established, the whole problem of surgical treatment changes. One must be meticulously careful in selecting the time for operation and the type of anæsthesia to be used. The nature of the operation to be performed is altered and comes well into the class of major operations. The immediate post-operative reaction and treatment are a source of anxiety to the surgeon and nurses, and the prognosis becomes somewhat problematical.

Primary toxic goitre, or acute Graves's disease, is a disease of young people, and for a minimum period of six months is essentially a problem for the physician.

Surgical treatment is better considered when the patients have reached a stage of some chronicity in the disease, unless by happy chance they prove to be among the few who may be cured by deep X ray therapy, removal of toxic foci and implicit obedience to a strict medical *régime*. The economic factor seems to be the great stumbling block in the treatment of these patients; they are not suitable for in-patient hospital treatment for the full period, and their home environment is frequently unsuitable, so that on occasions one is tempted to subject them to early operation. These are the only cases in which I have ever seen any good result from X ray treatment.

The age periods in which surgical treatment is usually undertaken are the early thirties and the late forties. The patients in the former group are mostly the uncured residue of the primary cases, but we see a proportion of the toxic adenoma type. In the late forties we see chiefly the secondary types associated with multiple adenomata.

There is no doubt that the menopause plays a big part in precipitating an acute exacerbation of what has been a mild degree of toxicity. Some of the worst operative risks are encountered in patients with large diffuse goitres of acute onset, and next to these I put the people with large flabby thyreoids, the site of multiple adenomata in the middle-aged.

In preparing patients for operation one should insist on their being kept in bed in hospital in as quiet surroundings as possible. A four-hour chart is kept, and if there is any auricular fibrillation it is wise to get the nurse to try to record the apex beat rather than the pulse, and to enlist the cooperation of an expert physician. It is as well to remember that digitalis in these cases loses much of its potency in the control of auricular fibrillation; but it is as well prior to operation to give two or three large doses of digitalis to tide the patient over the first twelve hours or so.

Iodine is given in small doses, for example 0·18 to 0·3 cubic centimetre (three to five minims) three times a day after food, and if the patient does not react favourably to this it may be supplemented by quinine hydrobromide, also some mild alkaline mixture with a little bromide. On occasions "Luminal" or some such drug is used in addition, according to the preponderance of nervous manifestation; but be extremely careful with barbiturates in these cases and do not use them for immediate pre-operative medication. A full and generous diet is maintained, with plenty of milk, vegetables and fruit. If the patient's general condition is poor, some "Radiostoleum", "Ostelin" or other preparation containing vitamin *D* is given with meals.

I wish to draw your particular attention to the necessity for giving alkalis in these cases in pre-operative as well as in post-operative treatment. As you know, the metabolic rate is always increased and the patients are living in a condition of general nervous hypertension. This means a heavy strain on the normal metabolic processes, with a corresponding increase in waste products; in other words, there is an increase in the acid content of the blood and other tissues, causing a big drain on the alkali reserve, so that the patients approach a condition of true acidæmia. If they are subjected to general anæsthesia and operation in this state, there is a grave risk of a fatal crisis developing with a real acidæmia. I feel sure that this was a big factor in those severe post-operative reactions we used to see before the necessity for careful pre-operative treatment was recognized.

I generally ask for a basal metabolic rate estimation before this pre-operative treatment is instituted, and in ten to fourteen days another estimation is made. If patients react favourably it will be manifest clinically. They feel rested, lose their sense of weakness, sleep better, and their pulse rate reaches a stable level, generally 20 to 30 beats per minute less than it was at first. This is due to fall in the metabolic activity and is confirmed by a lowered basal metabolic rate. Once this stable level is reached operation should be undertaken, for if this favourable time is allowed to pass the patients begin to show signs of relapsing and it is then difficult to restore their stabilized condition.

The hyperiodized patient is a problem. I think it is best in this condition to rely on alkalis, sedatives, rest and diet for four to six weeks and then to resume giving minimal doses of iodine. If they do not respond, a course of deep X ray treatment may help to lessen their toxicity to a sufficient extent to justify at least ligation of the superior thyreoid vessels under a local anæsthetic. Later, further operation may be undertaken, preferably in two stages.

Since the advent of this efficient pre-operative treatment I have practically given up the use of local anæsthesia. I find that premedication with plain morphine, 0·008 to 0·01 gramme (one-eighth to one-sixth of a grain) by hypodermic injection one and a half hours prior to operation, and a suitable dose of paraldehyde by rectum half an hour

later, followed by a gas anæsthesia, gives just as good results and allows the surgeon greater freedom.

A few words about paraldehyde. This is undoubtedly the safest of all drugs for premedication, and for toxic goitres in particular. It has a wide margin of safety, its dosage can be easily and adequately estimated, it does not make the patients helpless afterwards, and it gives them a good thirst, so that they will take plenty of fluids even before consciousness is regained, as they are quiet and docile, and will swallow readily.

The time factor is very important, and I feel that unless the operation can be completed within forty-five minutes it is wiser to do it in stages. I would urge surgeons beginning thyreoid surgery to bear this in mind and to plan the operation in two stages until they achieve the manual dexterity and operative technique which will enable them to embark on the complete operation. Very occasionally with bad risks a preliminary ligation of the superior thyreoid vessels will help. Ligation also gives one some idea of the reaction likely to occur after thyreoidectomy; but be sure to ligate the veins as well as the arteries.

Now we come to the common question as to how much tissue to remove. In order to get down to a definite basis I have for many years removed the whole right lobe, isthmus, pyramidal lobe, if present, and varying portions of the left lobe, depending on (*a*) the toxicity of the condition, (*b*) the size of the gland, (*c*) the age of the patient, and (*d*) the difficulty of access. As a rule it is necessary to remove more of the gland from males than from females, from the very toxic patients, from those with the multiple adenomatous type of goitre, and from those with severe cardiac disturbances. In fact, in the latter I feel that a practical ablation of the gland is the best thing to do. In all cases it is better to take too much than too little, as any tendency to myxœdema can easily be controlled by a judicious daily dose of thyreoid substance, whereas any persistent toxæmia threatens the individual with ultimate breakdown just as surely as the original condition, even if more slowly.

I cannot leave this question without again quoting Dunhill:

> No surgeon should operate on toxic goitres who is not prepared to remove enough thyreoid tissue to effect a cure, otherwise he will have sufficient bad results to render a serious dis-service to surgery.

On return to bed after operation the patients are given by the rectum glucose 10%, bicarbonate of soda 1%, and saline solution. Sufficient morphine is given to control restlessness; they are given as much fluid by mouth as they will take, and, if necessary, digitalis, digoxin, "Coramine", or quinidine after consultation with a physician.

One point as regards the operation: handle the gland as little as possible until the vessels of the upper pole have been ligated; then, grasping the upper pole in the thumb and forefinger of the left hand, gradually roll the gland down and towards the mid-line; this constricts the vessels and, I believe, prevents the forcing into the blood stream of large doses of toxic secretion.

Objections to operative treatment are often present in the minds not only of patients but of their doctors, and, as far as I can determine, are as follows:

1. The hope of some cure other than by operation.
2. The dislike of removal of such an important organ as the thyreoid gland, especially since we have no exact knowledge as to the ætiology of thyreotoxicosis.
3. The risks of operation. In competent hands, and including all types of cases, this is nowadays about 2%, a good figure, considering that ten years ago it was about 16%, and we now seldom refuse operation to any patient.
4. The scar. If the incision is made transversely in the lines of natural cleavage, if the deep fascia is sutured and the skin edges are approximated with interrupted stitches of unabsorbable material to be removed on the fourth and fifth days, the average scar is scarcely visible in three or four months.
5. Injury to the inferior laryngeal nerves. This occurs even in the best of hands in 1% or 2% of cases, but unless the nerve is actually severed it recovers its function in from two to six months and the risk of a permanently damaged voice is very small indeed. Such injury is most likely to occur when dealing with the small, hard thyreoids.
6. Incomplete cure and tendency to relapse. This depends chiefly on the experience of the surgeon, and I would again refer to Dunhill's remarks, already quoted, on this point. In a few of the young people there is at times a tendency for the portion of gland left at operation to hypertrophy, with some recurrence of the old symptoms. If this happens, a few treatments by X rays adjust the balance in most cases and obviate the necessity for further operation.
7. The standard of cure. One aims at restoring the individual to such a degree of normality that he or she can return to an active life and remain without periods of invalidism attributable to this particular disease. Of course, with damaged hearts this cannot always be attained, nor will severe exophthalmos ever disappear. The best standard of cure I have had is acceptance by the Australian Mutual Provident Society as "first class".
8. The possibility of other trouble supervening. The chief worry is that of putting on weight. I always say this is a good sign up to a certain point, but if excessive weight is put on, the basal metabolic rate should be estimated and it may be found that the patient is suffering from hypothyreoidism. If this be the case, the condition can be controlled by judicious dosage with thyreoid substance.

Hypoparathyreoidism.

One real difficulty has been met with, and that is the onset of hypoparathyreoidism. It is evidenced by a feeling of intense weariness and exhaustion, a

ILLUSTRATIONS TO THE ARTICLE BY DR. LEONARD C. E. LINDON.

FIGURE I.

Mrs. R.N., taken after injection of 14 cubic centimetres of "Thorotrast"; showing the filling of the internal carotid and the aneurysm (indicated by arrow) projecting backwards from the artery and arising shortly before the bifurcation of the artery.

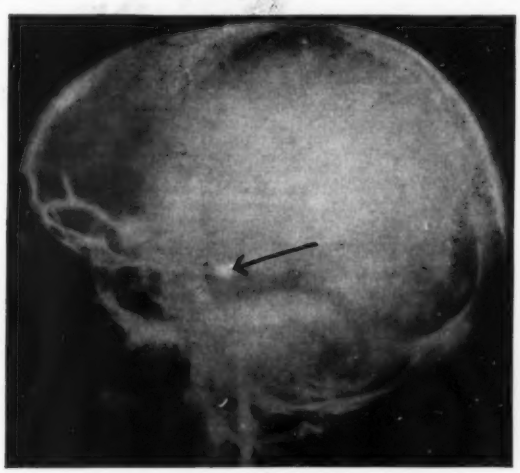


FIGURE II.

Mrs. R.N., taken four seconds later. The arrow indicates the residual "Thorotrast" in the pouch of the aneurysmal sac. The trunk of the carotid is still filled with "Thorotrast", but the dye has been swept on out of the remainder of the arterial tree.

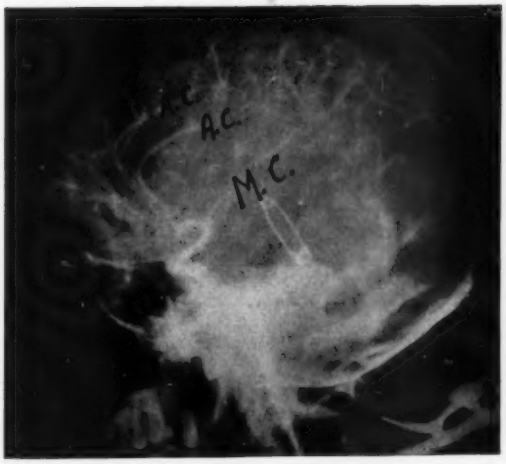


FIGURE III.

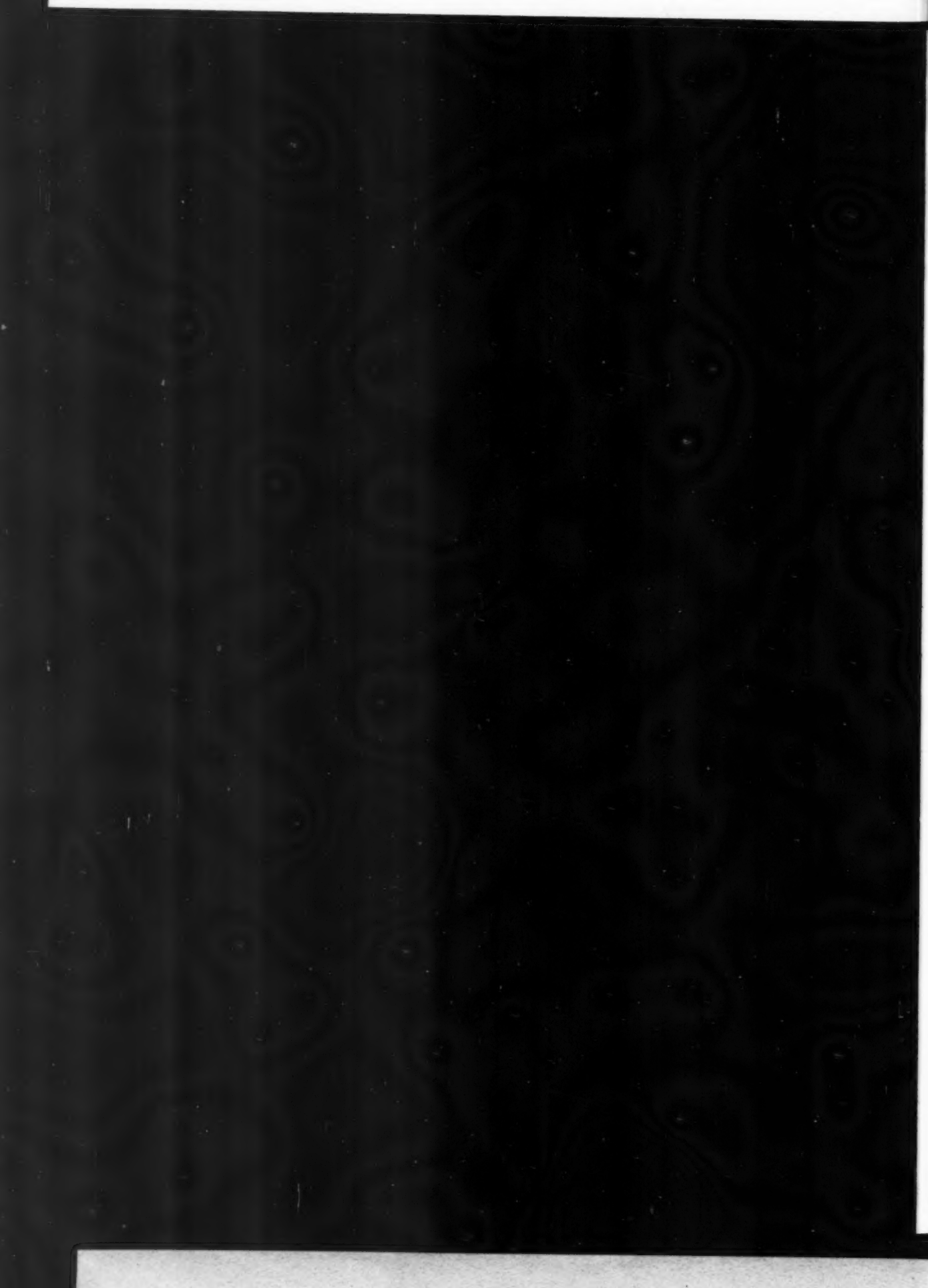
A.F.—right arteriogram. The carotid is well filled up to the circle of Willis, and the middle cerebral (*M.C.*) and both anterior cerebral (*A.C.*) arterial patterns are clearly seen. By comparison with the figures in Moniz's book these would appear to be quite normal.

FIGURE IV.

A.F.—left arteriogram. This shows a very much smaller internal carotid, which does not fill well up to the circle of Willis, the anterior cerebral arteries are not definitely seen, and the middle cerebral artery (*M.C.*) is represented by an abnormal network. Two large cortical veins (*V.*) are seen filled.

ILLUSTRATION TO THE ARTICLE BY DR. RICHARD FLYNN.

Urethrogram after injection of sodium iodide.



peculiar firming and stiffness of the muscles, often actual cramps, and in extreme cases tetany, swelling and tenderness of the breasts and, later, the development of follicular cysts of the ovary with menorrhagia and menstrual upsets. I have seen this condition develop as a result of X ray treatment, and one of the most difficult patients I have had to treat was a woman who had the condition prior to operation and as a result of X ray treatment. If the condition is secondary to operation, it is as a rule transitory and yields in two or three months to the giving of large doses of vitamin *D*, injections of parathormone, and possibly the giving of parathyreoid extract, although I have very little faith in the last mentioned.

Sometimes I am asked what is the result in those who are either unable to afford the luxury of complete invalidism and medical treatment or who do not respond to it and at the same time refuse the aid of surgery. My reply is that there are several possibilities: (i) They become burnt-out wrecks, with more or less permanent invalidism. (ii) auricular fibrillation and congestive heart failure close the picture. (iii) Acute mania develops. (iv) Glycosuria ensues. (v) Death results from general exhaustion. (vi) A fortunate few may establish a state of equilibrium and tolerance to a chronic toxæmia, such as will permit them to lead a shortened life of restful ease.

In conclusion, although the whole question of the proper treatment of this condition is still in the melting pot, I am sure that in the light of our present knowledge the quickest and most certain method of restoring the economic value of the sufferers and of bringing them as near to the normal as possible is by means of operation, carried through with adequate pre-operative precautions, strict attention to anæsthesia and operative technique and intelligent anticipation in the immediate post-operative treatment. An efficient follow-up system, by means of which final results may be definitely determined, is the only way of checking the efficacy of treatment, and should be instituted by all those undertaking surgery of the thyreoid gland.

THE RATIO OF LARGE TO SMALL LYMPHOCYTES IN PERSONS EXPOSED TO A LEAD HAZARD.

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IT has usually been regarded that the examination of white blood cells gives little information of value with respect to lead poisoning; but in a recent highly suggestive and interesting paper by A. E. Ferguson and T. Ferguson[1] it has been shown that the ratio of large mononuclear lymphoid cells to small lymphocytes has a closer relationship to the clinical condition than the stippled cell count, which has so far been regarded by most authorities on lead poisoning as of prime importance.

The above-mentioned authors carried out their investigations on shipbreakers who were subjected to a very great hazard (caused by oxy-acetylene flames impinging on lead paint) and who on the whole showed high stippled cell counts in films stained by Leishman's stain (500 to 25,000 per million). They found that shortly after beginning work on shipbreaking there was an increase in the above-mentioned ratio in the blood of the workers, and that so long as this ratio remained above 2:1 in any particular case, there was very little danger of the subject's showing signs of lead poisoning. If the ratio fell below 1:1, lead poisoning, if not already present, was imminent. Moreover, in the case of a worker showing a low ratio (say below 2:1) transference from a more severe to a less severe hazard or from a hazard to absence of hazard, as by going on holidays, was accompanied by an improvement in clinical condition and a concomitant increase in the ratio.

They regard the periodical determination of this ratio in persons exposed to a lead hazard as a more useful means of preventing the incidence of lead poisoning than determination of the stippled cell counts.

The hazard at the workings of Mount Isa Mines, Limited, is not now of such a severe nature as that associated with shipbreaking; but it was considered advisable to investigate the matter for several reasons.

Stippled cell counts depend to a considerable extent on the type of stain used, the technique of staining, and on the visual acuity of the observer, whereas the determination of the ratio of large to small lymphocytes is much less dependent on personal factors or on technique.

While it is probably true that an industrial plant in which the workers show an average stippled count of 10,000 per million will have a greater relative number of lead poisoning cases than one in which the average count is only 1,000, it is by no means true that the stippled count is always an index of the severity of the individual case.

It has been frequently observed at Mount Isa that an improvement in clinical condition is associated with a marked increase in the stippled cell count. This has already been pointed out by Ferguson and Ferguson.[1]

Any simple laboratory determination which might help in the diagnosis of doubtful cases or in the prevention of lead poisoning, is to be welcomed. The subject is of interest also from the point of view of hæmatology.

Standard of Size of Cells.

Ferguson and Ferguson[1] point out that the precise differentiation between monocytes and large lymphocytes may be difficult, and they therefore include both types in the classification large mononuclear lymphoid cells. This has been done in the present investigation also. They define large lymphocytes (and monocytes) as being at least the size of a polymorphonuclear leucocyte. This standard is rather vague, as some films have been

examined in which some of the polymorphonuclear cells were unusually small, although the majority were of normal size.

The standard of size adopted arbitrarily by the author has been 10μ. Cells equal to or greater than this in diameter are classed as "large", and those less than 10μ as "small".

Comparative determinations, using this standard and that of Ferguson and Ferguson, have given somewhat higher ratios in the former, in the proportion of 1·4 to 1.

Method.

The size was determined by means of an ocular micrometer and standard Zeiss millimetre objective graduated in divisions of 0·01 millimetre.

Slides were examined with an oil immersion apochromatic ×60 objective and ×10 eyepiece.

It is necessary to have the films one cell thick only. In thick films the leucocytes are frequently distorted in shape and apparent size.

There is usually a greater concentration of the larger leucocytes towards the edges of the films, and in order to get accurate counts the films must be examined in strips at right angles to the long axis, and completely from edge to edge of the film, commencing at the mid-line and finishing there, or else commencing at an edge and finishing at the other edge.

At least 50 lymphocytes were counted in each film.

Leishman's stain was used in a concentration of 0·15 gramme (Burroughs Wellcome "Soloid") in 100 cubic centimetres of pure methyl alcohol. The film, after air drying, was stained for one and a half minutes, the stain then diluted with twice the volume of distilled water and the diluted stain left on for a further ten minutes. The film was then washed in distilled water and dried.

Ratios in Persons Not Exposed to Lead Hazard (Class A).

In order to determine the value of the ratio of large mononuclear lymphoid cells to small lymphocytes in healthy persons not exposed to lead hazard, twenty-five healthy persons who had no known history of exposure to lead hazard were examined. They included workmen on construction of the new Mount Isa District Hospital, the hospital staff, and applicants for work at Mount Isa Mines, Limited. The average value of the ratio was 1·2:1.

Ratios in Persons Exposed to Lead Hazard but Not Showing Symptoms of Lead Poisoning (Class B).

The group of persons exposed to lead hazard but not showing symptoms of lead poisoning was made up of employees of Mount Isa Mines, Limited, from whom routine films were obtained, or who applied for examination to the author, but were fit for work. That they were exposed to a definite hazard was shown by the stippled cell count or a significant concentration of lead in the urine, or both.

The average value of the ratio in these cases was 3·3:1.

Ratios in Persons Suffering from Compensatable Lead Poisoning (Class C).

In 33 cases of compensatable lead poisoning the average value of the ratio was 1:1. In the 13 more severe cases the average value of the ratio was 0·84:1, and in the 20 less severe cases the value was 1·1:1. The distribution of the values of the ratio in these two classes is shown in Table I.

TABLE I.
Ratio of Large Mononuclear Lymphoid Cells to Small Lymphocytes.

Range of Values of the Ratio.	More Severe Cases.		Less Severe Cases.	
	Number.	Percentage.	Number.	Percentage.
0·0 to 0·49 ..	3	23	1	5
0·5 to 0·99 ..	7	54	5	25
1·0 to 1·49 ..	3[1]	23	11	55
1·5 to 2·0 ..	0	0	3[2]	15
Total	13		20	

(The average value of the ratio in the more severe cases was 0·84 and in the less severe cases 1·1.)

[1] In two of these cases, with values of the ratio 1·45 and 1·48, respectively, blood films were not obtained until some days had elapsed after the commencement of treatment. In all probability the ratios were much lower when the patients first reported.

[2] One of these patients was not examined until three months after exposure had ceased.

The more severe cases are associated with lower values of the ratio and less severe cases with higher values. In 77% of the more severe cases the value was below 1, whereas in 70% of the less severe cases the value was between 1 and 2. The percentage distribution of the values of the ratio in the three classes of subjects is shown in Table II. This table

TABLE II.
The Percentage Distribution of the Values of the Ratio.

Range of Values of the Ratio.	Class A.		Class B.		Class C.	
	Number.	Percentage.	Number.	Percentage.	Number.	Percentage.
0·0 to 0·99	7	28·0	3	7·3	16	48·5
1·0 to 1·99	18	72·0	6	14·6	16	48·5
2·0 to 2·99	0	0·0	14	34·1	1	3·0
3·0 and over	0	0·0	18	43·9	0	0·0
Total ..	25		41		33	

The average value of the ratio in Class A was 1·2, in Class B 3·3, and in Class C 1·0.

illustrates clearly that exposure to a lead hazard and consequent absorption of lead result in an increase in the ratio. When the value of this ratio, during continuance of exposure to the hazard, falls below 2, disabling symptoms may be expected, if not already present.

Even after removal from the hazard for a considerable period, mobilization of stored lead may occur, with the appearance of symptoms or exacerbation of symptoms already present and a concomitant fall in the value of the ratio.

In the case of subjects suffering from compensatable lead poisoning the higher values of the ratio which develop after removal from the hazard and

appropriate treatment may persist for some considerable time owing to the effect of the stored lead. If the subject does not return to the hazard and the case has not been a severe one, the ratio drops below 2 even while the subject is well. This is due to the return of the subject's condition to that of a person not exposed to a lead hazard.

With regard to other effects of lead absorption on the leucocytes, a definite relative lymphocytosis and a shift to the right in the Arneth count have been frequently observed in this investigation. These facts have already been reported by Ferguson and Ferguson.

The significance of the increase in the ratio on exposure to lead and the fall which is associated with deterioration in the clinical condition are not known.

Ferguson and Ferguson quote Russell[2] as suggesting that the bone marrow reacts to small doses of lead and throws out punctate red blood cells, but that if the dose is increased above a certain quantity it becomes paralysed and basophile cells disappear from circulation. The same may apply with respect to the reticulo-endothelial system and the lymphoid cells. In this connexion it is worth noting that Rivera[3] has shown that secondary radiation from metallic lead stimulates the growth of *Penicillium*.

There is the possibility also that the increased ratio of large mononuclear lymphoid cells to small lymphocytes is of the nature of a defensive reaction, as suggested by Ferguson and Ferguson.

It is proposed in another paper to consider the relationship of stippled cell count to the ratio here discussed, and of both of these to the urinary lead concentration.

Summary.

Absorption of lead causes an increase in the ratio of large lymphocytes *plus* monocytes to small lymphocytes.

A fall in this ratio below 2:1 while the subject is exposed to the hazard is associated with definite symptoms of lead poisoning, usually of sufficiently severe nature to cause incapacity.

Generally speaking, the more severe the case, the lower the ratio.

The magnitude of this ratio is more closely associated with the clinical condition than is the stippled cell count.

The magnitude of this ratio is a simple and very useful indication by which to judge of the imminence or otherwise of lead poisoning and is an aid to diagnosis.

Acknowledgements.

The author acknowledges with thanks the provision of laboratory facilities by Mount Isa Mines, Limited, during part of the investigation, the cooperation of Dr. S. P. Barnett and Dr. E. J. Ryan, of Mount Isa Mines, Limited, by supplying a number of blood films, and the interest shown by Dr. F. F. Pincus and Dr. P. N. Grieve in the investigation. He also wishes to thank Dr. N. C. K. Lane, Principal Medical Officer of the State Government Insurance Office, for drawing his attention to the paper by Ferguson and Ferguson, and those people who supplied samples of blood.

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CEREBRAL ARTERIOGRAPHY.

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INTRODUCTION.

SINCE 1930, Professor Egaz Moniz and his assistant, Dr. Lima, of Lisbon, have been bringing to perfection their work on cerebral angiography, using "Thorotrast" as their opaque medium. Moniz, in a recent publication (*"L'Angiographie Cérébrale"*, 1934), has described in detail his most ingenious radiological technique, whereby films are taken at intervals of one second up to six seconds, by which time they have obtained radiographic records of the pattern of both arteries and veins within the cranium. As a rule, within ten seconds all of the opaque substance has passed out of the intracranial vessels.

The writer had the privilege last year of being present at demonstrations by both Moniz and Lima, and of seeing the application of their work to neurosurgery by Norman Dott, of Edinburgh. In order to make full use of this method of diagnosis of intracranial tumour it is necessary to have the use of Moniz's radiological apparatus. But Dott has shown that with the usual radiological equipment it is possible to diagnose and localize with certainty certain vascular abnormalities, such as intracranial aneurysm and angioma. And the following short series of cases is considered worthy of publication, because of the increasing frequency with which the conditions of spontaneous subarachnoid hæmorrhage and intracranial aneurysm are being suspected and diagnosed. Few of the recent achievements of neuro-surgery can equal Dott's application of Moniz's work in the relief of a condition which in itself is benign, but which, without adequate treatment, generally recurs until the final fatal hæmorrhage occurs. The intravascular injection of "Thorotrast" has been used many thousands of times—Löhr alone reported a series of one thousand cases—and no ill-effects have been observed. With the interested cooperation of the radiological staffs of the Adelaide Hospital and the Adelaide Children's Hospital it has been found possible to obtain in each case two skiagrams, one taken at the moment when 15 to 18 cubic centimetres of "Thorotrast" have been injected, and a second one either three or

four seconds later. Many interesting points have cropped up during the performance of arteriography in the few cases in this series and, as more cases are investigated, it is hoped that surgery will be increasingly used in an endeavour to prevent the fatal hæmorrhage.

The writer's interest in this subject was first aroused by Dr. E. Britten Jones, who had under his care in the Children's Hospital a child who suffered from repeated headaches and vomiting, with recurrent attacks of right third nerve palsy. The case is reported more fully below. Moniz claims, and supports his claims by admirable films, to be able to differentiate radiologically between angioma, glioma and meningioma, from the vascular patterns observed. This can probably be done with certainty only if his apparatus is available.

For many years it has been known that the commonest cause of a spontaneous subarachnoid hæmorrhage is the leak of blood from an aneurysm in the cerebral arterial tree; and the examination of *post mortem* material has made it obvious that these aneurysms may be divided into two main groups: (i) those due to developmental weakness in the walls of one of the arteries, most frequently at or just proximal to the bifurcation of one of the larger vessels; (ii) an acquired structural defect in the wall of an artery associated with the degenerative changes of atheroma.

Of these two groups, the former is met with in children and adults in the first half of life and the latter group in middle or old age, and evidence of arterial disease will be found elsewhere in the body. The advance in this work of Moniz and Dott lies in the fact that diagnosis and successful treatment can be carried out in this group of young patients, in whom conservative measures cannot prevent an ultimate and fatal hæmorrhage.

Ligation of the internal carotid artery at any age is liable to result in grave interference with the blood supply of one hemisphere; and of course the risk increases with the age of the patient. Dott states that in his opinion ligation should not be performed merely because the patient has had one subarachnoid hæmorrhage, unless the hæmorrhage has been a severe one with advanced neurological signs, and unless the aneurysm can be definitely localized. But he considers that it is justifiable to risk the possible transient hemiparesis if the patient has recurring hæmorrhages.

Reports of Cases.

Case I.

L.H., a female, aged twelve and a half years, was under the care of Dr. E. Britten Jones at the Adelaide Children's Hospital. Her history dates back to 1930, when, at the age of seven years, she was brought to the Adelaide Children's Hospital with the complaint that for eighteen months she had been subject to severe vomiting attacks. In that period she had had about twenty such attacks, which had lasted as long as two days. The vomiting attacks were associated with pain in her right eye. The child was brought to the hospital because during the last attack the pain in the right eye had been more than usually severe, the right eyelid had drooped, and the child appeared to be squinting.

When examined in December, 1930, the only abnormalities noted were right ptosis and dilatation and inactivity of the right pupil, with some slight paresis of the external ocular muscles of the right eye. The fundus was normal. There was no reaction to the Mantoux and Wassermann tests; the spleen was palpable. X ray examination of the skull showed no abnormality. In spite of the negative result of the Wassermann test she was suspected of being a subject of luetic cerebral disease and was treated by mercurial inunctions, and her condition gradually improved. She was readmitted in October, 1935, as by this time Dr. Britten Jones suspected the probability of an intracranial aneurysm. The history since 1930 was that she had suffered from many bilious attacks, with frequent vomiting and pains in the right eye. The right eyelid was still drooping and there was still evidence of partial third nerve palsy. No other neurological abnormalities were found. Lumbar puncture was performed and the fluid was clear and colourless; there was no increase of globulin or total protein and no increase of cells. She recovered from this condition and was readmitted in February, 1936, for the purpose of arteriography.

On February 27, 1936, under ether anæsthesia, an endeavour was made to perform arteriography. The bifurcation of the right carotid artery was exposed and twelve cubic centimetres of "Thorotrast" were injected; unfortunately, a considerable amount of this escaped from the wall of the artery and the photography had to be repeated after the injection of a further eight cubic centimetres. The resulting films were not absolutely conclusive. The internal carotid had a definite fusiform enlargement between the *foramen lacerum medium* and the anterior clinoid process, and an abnormally serpentine course, and the skiagram taken three seconds later revealed a retention of "Thorotrast" in this part of the artery. It seemed that the films were not sufficiently conclusive to justify immediate ligature of the artery. This investigation was performed during one of the intervals between her attacks of third nerve palsy, and it was decided that it would be wiser to await the next attack and repeat the arteriogram.

Owing to faulty injection, the resulting films were indefinite; but there does appear to be a fusiform enlargement of the carotid in the region where such enlargement would implicate the third nerve; and the later film shows a residue of "Thorotrast". This investigation was performed at a time when the third nerve showed no abnormality. If she returns with another attack of ptosis *et cetera*, another arteriogram may confirm the original impression. In this situation an aneurysm would be unlikely to give rise to xanthochromic discoloration of the cerebro-spinal fluid, as the vessel is still extradural and there was no question of rupture of a vessel.

Case II.

Mrs. S., aged forty, was under the care of Dr. C. T. Pitcher. For at least four years she had complained of intermittent attacks of intense headache and queer "turns". At the beginning there was nothing particularly distinctive about the headaches or the "turns", except that she felt a strange giddiness and the feeling that she was turning round. As time went by she used to feel that she would fall down in the attack of headache. Two years ago she was seen in consultation by two ophthalmic surgeons and there was some uncertainty as to whether the appearance of her fundus was due to hypermetropia or to an early stage of papilœdema.

The headaches gradually got worse and became more severe in degree and sudden in onset, taking frequently several days in bed before relief was obtained. Neurological examination still revealed no abnormal signs. She

had not been feeling well for several months, but could not describe her feeling any better than by saying that she felt certain there was something wrong in her head.

On January 13, 1936, she was seized with the most intense headache in the left side of the forehead, the left eye, cheek and left side of the neck; she was seen in the attack by Dr. Pitcher, who knew the patient well and was convinced that her pain was extremely severe—as severe as that of a true tic, but in this case absolutely persistent. She could not move her neck in any direction without increasing pain. She was absolutely prostrated; she vomited repeatedly and gained some relief thereby. She was admitted to hospital forthwith, and with rest in bed the headache gradually cleared up. Her systolic blood pressure during the last two years has slowly risen from 100 to 155 millimetres of mercury.

On January 19, 1936, she began to complain of double vision, and it was noticed that she had drooping of the left eyelid. When seen on January 22, 1936, she limited all her complaints to her head and neck. The headache was still intense and it increased together with the onset of double vision; the headache was almost entirely limited to the left side of the forehead and the left eye and cheek, although there were some pain and stiffness in her neck.

The visual fields, when measured in 1933, were normal; but they had not been measured since. The optic disk was peculiar and it was considered by a consultant that she had advanced papillœdema in both eyes, but much more severe in the left; the visual acuity was normal. Dr. Pitcher found that both pupils were normal in shape, size and activity until the onset of ptosis, when the left pupil became dilated and inactive. When I saw her first the movements of the right eye appeared to be normal; there was complete paralysis of the left third and fourth cranial nerves, with complete ptosis, some proptosis and elevation of the left eyeball, and slight œdema in the left infraorbital region. No motor or objective sensory change was found in either fifth nerve. The remaining cranial nerves were normal. It seemed to me almost certain that the diagnosis was one of aneurysm of the internal carotid artery, in that portion of the vessel which is in relation with the cavernous sinus, rather than an intracranial tumour, which had been suggested by the papillœdema. I was not able to satisfy myself that papillœdema was present, and therefore I considered it reasonably safe to perform lumbar puncture. This was done on January 24, 1936. The pressure was 170 millimetres of water, and the fluid was clear and colourless, with a trace of globulin present and 20 milligrammes of protein per 100 cubic centimetres. There was macroscopic blood present, which must have been the result of the puncture. The absence of gross changes of the chemistry of the cerebro-spinal fluid seemed to me to confirm the diagnosis of aneurysm of the artery within the dural confines of the cavernous sinus, and I advised diagnostic arteriography, to be followed, if necessary, by ligation of the internal carotid artery. This advice was given reluctantly, owing to the possibility of some residual hemiplegia and aphasia; but it seemed that this risk must be taken in order to avert the possibility of sudden and fatal hæmorrhage.

On January 24, 1936, before the decision had been reached, she was given 0·01 gramme (one-sixth of a grain) of morphine to ease her headache (about ten hours after lumbar puncture). About half an hour later, when almost asleep, she complained of intense headache and then collapsed. Dr. Pitcher saw her within a few minutes and found her to be in a condition of acute pulmonary œdema, with a systolic blood pressure of only just 100 millimetres of mercury. He found no further neurological signs. Next morning she complained of seeing brown spots in front of her right eye, and examination revealed several retinal hæmorrhages in the right eye, but none in the left. Interference was withheld while she recovered from her pulmonary condition. By January 29, 1936, the ocular palsy was as before; but there was marked hyperæsthesia in the region of the first division of the left fifth nerve.

On January 31, 1936, an attempt was made to obtain an arteriogram; but this was our first effort, and faults in technique, both in surgery and radiology, resulted in unsatisfactory photographs. The injection of "Thorotrast" was made into the left internal carotid artery under local anæsthesia, the artery itself being very sensitive. The second injection was made later in the day, but it was made too slowly and the resulting film again was not a success. The patient noticed no ill-effects from the injection; but it was of interest to note that the injection of 15 cubic centimetres of "Thorotrast" under pressure into the internal carotid caused an acute access of pain in the area of the first division of the fifth nerve. The internal carotid was temporarily occluded by a silk sling, and the cessation of blood flow in this artery appeared not to interfere with her at all; she continued to talk and to move her right arm and leg freely when asked to do so. This occlusion was maintained for upwards of five minutes, and the failure of any effect was regarded as being in support of the diagnosis, as indicating that the bulk of the supply of the circle of Willis was being provided by the opposite carotid and basilar arteries. Therefore the left internal carotid artery was finally doubly tied with silk and the wound closed. Improvement was very gradual and the headache, though less intense, persisted for nearly four weeks, but has now entirely cleared up. The ophthalmoplegia also has diminished very slowly; but if one's conception of the anatomy and pathology of this condition is accurate, one could not expect a rapid recovery of her third nerve. At no time since ligation of the artery has there been any suggestion of interference with the blood supply of the brain. Lumbar puncture on February 17, 1936, showed the cerebro-spinal pressure to be 125 millimetres of water and the faintest suggestion of xanthochromia.

When seen again on March 24, 1936, the patient had made a good recovery; the ophthalmoplegia on the left side had almost entirely cleared up and the pupil had become normal in size and in reactions. The eyelid still drooped a little. Considering that the *levator palpebræ superioris* was paralysed for a period of at least four weeks, an earlier recovery might have been obtained by a temporary suture of the upper lid to the right eyebrow, in order to place the paralysed muscle in a position of relaxation. An endeavour was made to do this by means of strapping; but the suture would have been more effective.

Case III.

Mrs. R.N., aged thirty-three years, was under the care of Dr. Guy Lendon. In the previous few months she had suffered from severe bouts of intense headache in the area of the first division of the right fifth nerve. In some of these attacks she complained of backache and she was thought to have sciatica. During the past ten days one very severe headache was followed by a convulsion and unconsciousness, with residual left hemiparesis, with very variable physical signs but definite left extensor plantar reflex and the loss of the left superficial abdominal reflex. Forty-eight hours after this particular attack the cerebro-spinal fluid was xanthochromic. There was no reaction to the Wassermann test.

When seen on February 22, 1936, she was complaining of agonizing headache in the area of the first division of the right fifth nerve; this area was hyperæsthetic. There was almost complete ptosis of the right upper eyelid, with the right pupil larger than the left. The activity of the right pupil varied with the degree of her headache, and at times was dilated and inactive, but was always larger than the left. There was a very doubtful abducens palsy and, I thought, a very slight proptosis of the right globe. Hæmorrhages were present in the right retina; the ophthalmologist's report was that there was atrophy of the left disk with failing vision, and her visual fields, when tested by confrontation, showed loss of both lower quadrants of the left field. The other cranial nerves showed no abnormality. The systolic blood pressure was 120 millimetres of mercury. The signs of hemiparesis gradually cleared up.

The impression formed was that she was suffering from an aneurysm of the internal carotid artery within the cavernous sinus.

The headache seemed unbearable and was not greatly affected by 0·02 gramme (one-third of a grain) of morphine, even when repeated doses were given.

An arteriogram was taken under "Percaine" anæsthesia. The bifurcation of the right common carotid was exposed and the internal carotid was dissected free and encircled with a silk sling. Moniz records in his recent book that frequently better films are obtained by injecting into a closed circulation, that is to say, by stopping the flow of blood in the internal carotid just before the moment of injection. So in this case the carotid was temporarily occluded and 14 cubic centimetres of "Thorotrast" were injected. The first film was taken as soon as this point had been reached, and the second film was taken four seconds later. In the first film (see Figure I) the arterial system is well filled and it shows an elongated saccular aneurysm projecting backwards from the internal carotid artery just proximal to its bifurcation. (It is interesting to notice that once again, as in Case II, the injection of "Thorotrast" gave rise to severe pain in the region of her previous headache, the ophthalmic division of the right fifth nerve.) The second film (see Figure II) shows that the internal carotid artery, up to the anterior clinoid process, was still full of "Thorotrast", as the circulation was still being occluded in the neck; it also shows that the sac of the aneurysm was still full of "Thorotrast". But the "Thorotrast" had all been swept on, in spite of the occlusion of the right internal carotid, and I think it was logical to arrive at the conclusion that an adequate collateral circulation and the circle of Willis were already functioning. The right internal carotid was then ligated and the wound closed.

The relief of headache was dramatic; within an hour of operation they patient declared herself to be entirely free of headache, having suffered incessantly for the preceding three weeks. Admittedly she was an emotional and hysterical type of patient (and in fact four nights later became quite irrational, but recovered quite quickly the next day); but even that could not explain the remarkable cessation of pain and headache. The signs of ophthalmoplegia steadily cleared up and she was discharged from the Adelaide Hospital on March 13, 1936, in excellent health.

It is difficult to explain the loss of both lower quadrants of the left visual field or the left-sided optic atrophy. The position of the aneurysm was such as to have caused pressure upon fibres passing to the nasal half of the left retina, but it is difficult to explain the involvement of the fibres of the temporal half. But reports indicate that her sight in that eye is improving already and that her visual fields are almost normal. There have been no signs of hemiplegia since ligation of the artery, so that it is evident that the bulk of her cerebral supply was being carried on before the operation by the opposite carotid and the basilar artery.

Case IV.

Mrs. D., aged thirty-three years, was seen in consultation with Dr. L. Opit on March 6, 1936. Her history and physical signs were the most characteristic of all the patients in this series; unfortunately the disease progressed to a fatal result. The history obtained from her mother was as follows, and is quoted in detail. She was in good health until twelve days before, when she had sudden terrible headache in the right side of the forehead and the right eye. It eased off gradually while she was resting in bed. Four days later she had another sudden and intense attack in the same region; the pain also went down her back, but not to her legs. She fainted with the pain. She remained in bed two days and the headache passed off. Three days after the commencement of this attack intense right frontal headache recurred. Two days later headache was still present. There was ptosis on the right side; the right pupil was big and the eye was staring; there was diplopia. The patient was sent to hospital. The night before I saw her she had had a fit in the middle of the night. No details of the fit were obtainable. She had become conscious again by the morning. At 10 a.m. Dr. Opit did lumbar puncture. The fluid was under pressure and heavily blood-stained. During the lumbar puncture she said she felt a fit coming on; she had a generalized fit and lost consciousness. She had not regained consciousness at 6 p.m. She was having repeated general tonic spasms; her arms were fully extended and internally rotated; there was opisthotonos; the legs were in full extension and the ankles were fully plantar flexed. These spasms were accompanied by groaning and increased depth of respiration. She also had intermittent bouts of profuse sweating all over. There was incontinence of urine and fæces. The temperature was 37·7° C. (99·8° F.), the pulse rate was 64 to 84, and the respiration rate 10 to 20 per minute. The systolic blood pressure was 100 and the diastolic pressure 70 millimetres of mercury. There was right ophthalmoplegia (*interna* and *externa*). The left eye was normal. In the right fundus there was a swelling of the nasal margin of the disk (about 0·5 diopter), and there were many hæmorrhages (? subhyaloid). Both corneal reflexes were active; the right appeared less active than the left. The tendon reflexes were all hyperactive (++++) but symmetrical. The superficial abdominal reflex was absent on both sides. Both plantar reflexes were extensor.

It was considered that there must be an aneurysm of the internal carotid artery within or just above the cavernous sinus. It was still leaking, and the prognosis was considered to be very bad unless the artery was tied at once.

Dr. McLaughlin reported that the cerebro-spinal fluid showed an increase of protein up to 0·5%, much more than could be accounted for by blood alone.

At 8 p.m., under "Percaine" anæsthesia, the right internal carotid was doubly tied with catgut. The pulse rate was 125 per minute before operation and 110 at the end. The pressure of cerebro-spinal fluid at lumbar puncture was 320 millimetres of water; 10 cubic centimetres of fluid were run off, the last few cubic centimetres containing much more blood than the first few cubic centimetres. Pressure was reduced to 120 millimetres of water. The patient's colour was still very good.

On the morning of March 7, 1936, her condition seemed a little better; she had had no more of the tonic spasms since the ligation of her right carotid artery, her corneal reflexes were very brisk, and she roused to painful stimulation. In the afternoon her temperature was beginning to rise and her pulse rate had gone up to 140 per minute. An endeavour was made to perform lumbar puncture, and no cerebral-spinal fluid was obtained until suction was applied with a syringe, when about two cubic centimetres of cerebro-spinal fluid full of blood clot were obtained. As the spinal subarachnoid space had probably been occluded by a clot from the previous day's bleeding, a cisternal puncture was done and again only a little cerebro-spinal fluid could be obtained, consisting almost entirely of clot. Her condition continued to depreciate and she died late that night.

On looking back, one feels that the right thing to have done after the failure of the cisternal puncture would have been a suboccipital decompression, as it seemed certain that her death was due to compression by blood clot in the region of the *foramen magnum*. Unfortunately permission for *post mortem* examination could not be obtained.

Case V.

A.F., aged twenty-six years, an unemployed male, was under the care of Dr. S. R. Burston at the Adelaide Hospital. The history given on January 6, 1936, was that ten days previously, while in the lavatory, he experienced a severe stabbing pain all over the head, the pain being very intense. The pain gradually diminished and had disappeared entirely at the end of a week. On January 5, when riding a bicycle, he was seized with sudden severe

pain in the small of the back, and pain in the head and ears, and preexisting deafness was increased. He vomited at the same time.

He complained of intense pain in the head and back; marked photophobia was present. At that time the neurological examination revealed no abnormality except neck rigidity, bilateral middle-ear deafness, some stiffness in the muscles of his legs, and tenderness along both sciatic nerves.

On January 8, 1936, lumbar puncture revealed sterile cerebro-spinal fluid, opalescent and straw-coloured, under a pressure of 200 millimetres of water. The blood and cerebro-spinal fluid did not react to the Wassermann test.

His condition improved after lumbar puncture until January 28, 1936, when, while feeling very well, he suddenly had three epileptiform seizures, which consisted of loss of consciousness, tonic spasms and irregular respiration; the pulse rate was 90 to 100 per minute. The fits were followed by stupor for a quarter of an hour to half an hour. After the attacks the plantar reflexes were extensor and the right pupil was found to be larger than the left. Tendon reflexes in the right leg were more active than the left. On the next day he described an alteration of sensation in the right arm and leg, as though the limbs did not belong to him. No objective changes were found. Tendon reflexes on the right side were more active than on the left, and a Babinski reflex was present on the right side. The optic disks were normal. He continued in much the same condition for several days, and on February 3, 1936, lumbar puncture was performed, and the cerebro-spinal fluid was again found to be definitely yellow and under a pressure of 250 millimetres of water.

On February 7, 1936, the patient had become dull and disorientated; the temperature was again rising; he was irrational and incontinent of urine. The neurological signs were as follows: slight left lower facial weakness; the tendon jerks of the right arm and leg were more active than those of the left. Both plantar reflexes were flexor. Both pupils were active, but the left was definitely larger than the right.

The patient remained in this condition until February 26, 1936, when, under "Percaine" anæsthesia, eighteen cubic centimetres of "Thorotrast" were injected into the right internal carotid artery, with the circulation closed. An X ray film was taken at the end of the injection, and the second film, taken three seconds later, was spoilt by the fact that the patient had a fit just as the second exposure was made. The film showed the perfectly normal pattern of the right middle cerebral artery and both anterior cerebral arteries (see Figure III). There were no after-effects of the fit, except weakness of the lower part of the right side of the face.

By March 6, 1936, the patient was feeling very well and showed no abnormalities whatever except persistent weakness of the lower part of the right side of the face. The pupils were equal and active.

On March 14, 1936, a left internal carotid arteriogram was obtained; 18 cubic centimetres of "Thorotrast" were injected while the circulation in the artery was occluded, and the occlusion was maintained for over ten seconds. No fit or disturbance of any sort was noted. The films (see Figure IV) show that the left internal carotid is very much smaller than the right; there is no clear pattern of the anterior cerebral artery at all, and the middle cerebral artery is represented by a plexiform pattern resembling an angioma. The posterior parietal branch is very large. There is a definite and obvious asymmetry between the patterns of each side. It appears that the right carotid is supplying most of the brain and that the left side is abnormal, and is associated either with an angioma or some type of neoplasm, from which there has been slight leakage.

On March 20, 1936, the patient was very well and no abnormal neurological signs were discovered.

At present, from lack of experience, it is impossible to interpret the films with certainty. But from the fact that occlusion of the left internal carotid for ten seconds gave rise to no obvious signs of cerebral ischæmia, while occlusion of the right internal carotid after four seconds caused a generalized convulsion, and from the X ray evidence that the right artery is supplying the opposite anterior cerebral artery as well, it should be reasonable to infer that the circle of Willis is being almost entirely and adequately supplied by the right internal carotid and basilar arteries. I suspect that the appearance of the left arteriogram indicates an angiomatous malformation of the middle cerebral artery.

If another subarachnoid hæmorrhage should occur, then I believe it is justifiable to ligate the left internal carotid artery; otherwise it is extremely probable that the attacks will be repeated until a fatal hæmorrhage occurs.

Discussion.

Three cases showed retinal or subhyaloid hæmorrhages. All three showed extreme involvement of the nerves lying within the walls of the cavernous sinus, and it is probable that the swelling of the artery within the limited space of the cavernous sinus was sufficient to obstruct the flow in the retinal veins.

One other interesting problem arises. In the four cases in which arterial investigation was performed the internal carotid of the suspected side was completely occluded for periods of from five to ten seconds without producing any physical sign of cerebral anæmia; and, as has been recorded, occlusion of the artery on the normal side in one case (A.F.) for a period of less than four seconds produced a convulsion. This raises the question as to how long it is possible to occlude a normal carotid in a healthy young adult without interfering with the cerebral supply. Is it a matter of only one or two seconds, or do the relative contributions of the two internal carotids in normal individuals vary very much? And, as a more important corollary, is it reasonable to assume that if a suspected internal carotid artery can be completely occluded for at least ten seconds without producing any observable effect, then that artery can safely be ligated in the treatment of aneurysm? The experience—admittedly small—in two cases here recorded would seem to indicate that that is so. But information on this point would be welcomed.

No opportunity has yet arisen of performing a basilar arteriogram. Rupture of a basilar aneurysm is generally rapidly fatal. And, indeed, if one were radiologically demonstrated in time, the ligation of both vertebral arteries would have as ill an effect as the aneurysm.

Acknowledgements.

I wish to acknowledge my thanks to my colleagues herein named for their permission to publish notes of cases under their care.

Reports of Cases.

INTRAPERITONEAL RUPTURE OF THE BLADDER.[1]

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H.F., aged fifty-three years, a male, an inmate of one of the mental hospitals, was admitted to the Royal Prince Alfred Hospital on January 21, 1935, with the history that he had been kicked in the perineum three or four days previously by a fellow inmate and that he had passed no urine during this period. No attempts at catheterization had been made. He had been noticed to be ill, and much worse on the last day. He had vomited intermittently during the last two days and the vomitus was said to resemble coffee.

On examination it was noted that there was an extensive area of bruising around the perineum which had extended up over the pubis into the lower part of the abdominal wall, in the area in which extravasated urine is generally found. The abdomen moved freely and there was no dulness over the bladder, and the abdomen was not rigid. The scrotum was tensely swollen and blue; its dimensions were roughly 10·0 by 7·5 by 7·5 centimetres. In the perineum proper there was a tense blue swelling. He was given a hypodermic injection of morphine sulphate, 0·015 gramme (a quarter of a grain), and atropine, 0·6 milligramme (one one-hundredth of a grain), and operation was undertaken under nitrous oxide and oxygen. Through a low median line incision the bladder was exposed and was found collapsed. It was opened and some brownish material, which was presumed to be old blood and urine, was evacuated. On exploring the bladder, coils of small bowel were felt with the finger. An intraperitoneal rupture of the bladder was diagnosed and the peritoneal cavity was opened. About six pints of urine were sucked out with a sucker, and the rent in the bladder was exposed from the peritoneal side. This was sutured in the usual manner, and rubber drainage tubes were inserted into the bladder, peritoneal cavity and space of Ritzius.

The bladder was closed around the tube and the abdominal wound sewn up in layers. The patient was then placed in the lithotomy position and the swelling in the perineum was incised and much blood clot was evacuated. A catheter was passed through the penis and its tip appeared in the perineal wound. The urethral wall was identified and by the aid of a Teale's gorget the penile catheter was passed up into the bladder. No attempt to repair the urethra was made because of the condition of the patient and also because it was felt, on account of the inflamed condition of the urethral walls, that any sutures inserted would cut out.

One pint of saline solution was inserted into the peritoneal cavity before closure, and 500 cubic centimetres of 1·5% saline solution were given intravenously and two pints of normal saline solution subcutaneously.

Blood for estimation of blood urea and blood chloride was taken and some of the urine sucked out of the abdominal cavity was kept for determination of its nitrogen content. The biochemist reported as follows: Blood urea, 157 milligrammes *per centum*; the chloride content of the peritoneal fluid was 400 milligrammes *per centum*, and its urea was 900 milligrammes *per centum*.

On January 24, 1935, under nitrous oxide gas anæsthesia, the urethra was sutured around a catheter in its lumen, good approximation being obtained. The perineum was repaired. After operation the patient became very restless, and on February 3, 1935, he tore the whole perineal wound open and removed the catheters from the bladder and penis.

A further attempt at suturing the urethra and perineum was made on February 7, 1935, with satisfactory immediate result, but the patient again tore the wound open on February 15, 1935.

A third attempt at suturing the perineum was made on February 21, 1935, and the patient's arms were enclosed in a plaster cast to prevent his interfering with the wound. The perineal wound was soundly healed by March 4, 1935, and at weekly intervals subsequently, under "Evipan" anæsthesia, the penile catheter was changed. The urethra was syringed through with acriflavine and urethral bougies were passed. No difficulty was experienced in passing a number 30 French bougie.

On April 11, 1935, sodium iodide was injected with a glass syringe into the urethra, and a urethrogram (see Figure I) was taken. This shows that the urethra has soundly healed and that there is no sign of a perineal fistula.

The combination of a complete rupture of the urethra and intraperitoneal rupture of the bladder is unusual, as this is the only case in the records of this hospital. Yet the good result in this case as regards the ruptured urethra is due, in my opinion, to two things: (i) the lack of infection, and (ii) the intraperitoneal rupture of the bladder, as it prevented the irritation of the tissue spaces with extravasation of urine.

[1] The patient described herein was shown at a meeting of the New South Wales Branch of the British Medical Association on May 23, 1935.

Notes on Books, Current Journals and New Appliances.

THE MEDICAL ANNUAL.

POSSIBLY one of the most useful books that are published for the medical profession is "The Medical Annual". The volume for 1936 has been issued.[1] It is a worthy successor to those that have preceded it in the last fifty-three years. To those who do not know this book, it should be stated that it is a review of the progress in every branch of medicine during twelve months. The more important contributions to medical literature are reviewed by "contributors" of whom there are thirty-six. The editors are Dr. H. Letheby Tidy and Dr. A. Rendle Short.

To give an adequate idea of the scope of this book would require a great deal of space. It must suffice to point out that considerable attention has been given to the practical side of treatment—this is the intention of the publishers, for the sub-title is: "A Year Book of Treatment and Practitioner's Index". At the close of their introduction the editors write:

> As one looks through the material gleaned by our contributors from another year's medical literature, an outstanding impression is that there have emerged an unusually large number of comparatively simple methods of new treatment, for a great diversity of ailments, well worthy of the attention of every practitioner who desires to relieve his patients safely, quickly, and pleasantly.

We can do no better than repeat our previous recommendation that medical practitioners, particularly those in general practice, should form the "Medical Annual habit". If they do, they will never regret it.

[1] "The Medical Annual: A Year Book of Treatment and Practitioner's Index", edited by H. L. Tidy, M.A., M.D., F.R.C.P., and A. R. Short, M.D., B.S., B.Sc., F.R.C.S.; 1936. Bristol: John Wright and Sons Limited. Demy 8vo., pp. 624, with illustrations. Price: 20s. net.

The Medical Journal of Australia

SATURDAY, JUNE 20, 1936.

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THE PILOT, THE PUBLIC AND THE 'PLANE.

Most of man's activities, it has been well said, can be grouped together under one general heading, no matter how varied his occupations may seem. For the most part he is engaged in altering the relative position of matter at or near the earth's surface. In performing the manifold operations involved in this work man often faces perils to both bodily health and mind, a fact which has been responsible for the gradual rise in importance of preventive medicine and hygiene as protective measures. Almost every industrial enterprise has its peculiar dangers involving either the body or the nervous system. In this regard there seems no doubt that in the immediate future particular attention must be paid to the cause and prevention of the risks run by aviators.

For psychical reasons as well as for reasons of anatomical structure, man is not born to fly. Only after serious planning and the preparation of elaborate apparatus can he embark on long aerial journeys, or determine problems of flight which a flock of migratory birds could settle overnight. In man's mind, too, is the fear innate in all normal human beings — the horror of falling from a height. Few people, even not excluding some famous aviators, can look down the stair-well of a high building without qualms. This fear, in the case of aviators at least, may be hidden—repressed into the unconscious, as the phrase goes—but it struggles to be free or to divert itself into fresh channels, to sublimate itself. And in the mind of the aviator in this unhappy state is a further chaos of emotions: a dread of the scorn of his colleagues, a feeling that he is becoming inexpert in the technique of flying, and a dread that he may suffer reduction in pay, with consequent loss of social status and economic security. But day after day, whatever his apprehensions, the pilot must fly, and flying, not only in war-time, but in civil life, is the most dangerous of occupations. Few airmen fly long without themselves "crashing" or seeing others "crash". For them the air must sometimes seem peopled with demons, but they must still build upon a shaky foundation of fear a strong fortress of courage.

At one time the public regarded aviators, if not as plain lunatics, at least as misguided enthusiasts. The Great War came and pilots were looked upon as heroes; and heroes they were. The War once over, aviation gradually became almost a humdrum profession in the eyes of the community. This change of view somewhat lessened the self-esteem of pilots, but it caused public confidence in the new mode of travel to take an upward jump. But through these years, and all too often, the public trust and the confidence of flying men have been rudely jarred by terrible catastrophes. Famous pilots vanish for ever or are killed in every circumstance of horror. Aeroplanes disappear into the blue with passengers and crew, lose their ways in fogs and storms, or drop like plummets from the sky.

> . . . The tragedy came with appalling suddenness . . . the 'plane appeared to be flying splendidly, when before the eyes of the spectators, it seemed to stall and then dropped like a flash to the ground . . . there was a terrific explosion and flames burst from the petrol tank. For a few minutes the fire burnt with terrific intensity and reduced the 'plane to small wreckage . . .

Inevitably, as in all such accidents, the pilot was smashed to pulp and his body turned to carbon. But the newspaper report here quoted naturally makes no comment upon the mental effect of this

terrifying spectacle upon the spectators, nor upon the damage done to the cause of aviation.

We wish to discuss these matters from the medical point of view. No doctor can doubt that the profession of flying has aspects which gravely upset the equanimity of both the pilot and his potential passengers. A noted contribution of the aeroplane to the common lot of mankind is that it has made war a more disgusting business than ever. During the Great War large numbers of military pilots who managed to escape violent deaths in the field were invalided out of the various air forces. They had given way before the tremendous strain and had contracted neuroses which rendered them inefficient as fighting units. It is important to bear in mind, however, that these disorders of military pilots differed in no important clinical detail, though they differed in severity, from the functional neuroses associated with civil aviation. For these latter the covering term of "aeroneurosis" has now been adopted. Aeroneurosis first manifests itself by gastric distresses, fatigue of the higher mental centres, emotional instability, nervous irritability, and increased motor activity of the busy, snappish type. Within the past few weeks Harry G. Armstrong[1] has published a graphic description of the whole syndrome. In general, the affection is not one of young pilots. But somewhere in the age group twenty-five to forty years, and commonly after four years or so of flying, the symptoms insidiously begin. Since candidates for air training are rigidly examined both on entry and periodically thereafter, all chances of their suffering from constitutional disease are eliminated. It was once believed that a slender physique bore some relationship to the disease, but as very many aviators are of slight build, this is not now considered to be a factor of any importance. The material chosen for the making of pilots is of the best. The men chosen are devoted to duty, personally attractive in character, energetic, alert, and generous in their impulses. Gastric distress is complained of from one to four years before the recognition of the established disease, and is of the nature of "hunger pain", but unaccompanied by anorexia, nausea, vomiting or eructations. The patient later becomes irritable and bad tempered, at the same time resenting sympathy and resisting all suggestions that he is ill. There ensues a period of misgiving as to the future, of nights filled with heavings and tossings, or by nightmares about flying accidents. Inability to concentrate, inattention to any matter in hand, and black depression are plainly evident. The personality may become almost paranoid and the sufferer growls out obscure grievances about petty persecutions and the onerous duties assigned him. The balancing tests reveal unsteadiness, and the whirling tests an increase in nystagmus time. Itching eyelids, blurring of vision in the face of bright lights, and smarting of the conjunctivæ are common.

Such in brief are the symptoms of aeroneurosis. Few objective signs have so far been found. But it is known that in laboratory animals a reproduction of the conditions to which pilots are exposed can produce severe central nervous lesions. Amongst these are congestion, œdema, hæmorrhage, and even destruction of the brain. Pathogenetically, the disease would appear to spring from intense and long-continued emotional shocks, leading to exaggerated physiological reactions and depletion of the higher nerve centres.

The effective treatment of the condition is bound up with an early diagnosis. An aviator of several years' experience who gives evidence of suffering from a functional gastric disorder, irritability, great mental fatigue and increased motor activity should, on a first attack, be kept under close medical observation. At the hint of a second attack the patient should no longer fly an aeroplane, but should be given employment at "ground work". An essential ingredient of the treatment is that the remuneration for this work should be adequate, in order that an already over-strained nervous system should not be burdened with financial worries. It is likely that in no long time the supervision of these details will become an established part of medicine. Science is doing its utmost, by improvements in materials and aeroplane design and by the development of safety devices, to minimize the possibilities of disasters in the air and to foster

[1] *The Journal of the American Medical Association*, April 18, 1936.

public confidence. It is for medicine to assess and treat any physical or psychical deviations in the pilot. This may be no easy task. Only yesterday man was content to walk three miles in one hour. Now he flies three miles in one minute.

Current Comment.

CHLORAL HYDRATE.

CHLORAL HYDRATE was discovered by Liebig in the year 1842 and was introduced as a hypnotic thirty-seven years later. It is produced by the action of chlorine upon ethyl alcohol, and is a crystalline substance of unpleasant taste and smell, productive of a feeling of warmth in the mouth. Coming as it did at a time when the only reliable hypnotics in the Pharmacopœia were opium and morphine, the new drug seemed likely to fill a very useful place in therapy. But before long the impression gained ground that chloral hydrate was unsafe. Cases were recorded of medical men who passed from sleep into death after too liberal a use of the hypnotic, and in several trials for homicide it was proven to have been feloniously administered in beer. The famous Professor Tyndal died of an overdose of the drug. The net result of many unfortunate happenings of the sort was that chloral hydrate earned the reputation of being an extremely dangerous drug, especially in cases of cardiac disease. Most text-books of pharmacology and toxicology evince a positive dislike for it and issue solemn warnings about the dangers of an overdose. A great German toxicologist has said that chloral hydrate may have a fatal effect in amounts only double the amount of the maximum therapeutic dose of three grammes, and he cordially welcomes its replacement in the list of available soporifics by the less poisonous urea derivatives. Up to the year 1900 about 120 fatal cases of poisoning due to chloral had been reported. The first was that of Fuller (*The Lancet*, 1871), whose patient died thirty-five hours after the ingestion of only two grammes. Taylor is more reassuring; his opinion is that, while chloral is a dangerous hypnotic in cases of cardiac disease, it cannot of itself be considered to possess a high degree of toxicity. It may be presumed to act in a selective manner upon cardiac muscle, quickly affecting an unhealthy heart, but leaving a healthy one unaffected. The cerebral cortex, and particularly the *medulla oblongata*, may be depressed and ultimately paralysed, and the results upon the heart are said to be similar to those of chloroform.

It thus appears that the use of chloral hydrate has been roundly condemned by many competent authorities. On the other hand, it has had capable protagonists. Twenty years ago Gunn said that he could unearth no reliable evidence to convince him that the drug, given in therapeutic doses, had any depressant action upon the heart, and that further investigation was required before such a reputation was permanently associated with one of the most reliable of hypnotics. Stockman, an acute observer, expressed equally favourable views; and Balfour, of Edinburgh, was accustomed to prescribe for patients with pneumonia a mixture containing ten grains of chloral hydrate, combined with tincture of digitalis. Thus, he said, his patients were enabled "to sleep through their pneumonia".

Stanley Alstead[1] now stands forth as a champion for the hypnotic. He has lately observed its effects upon fifty-five patients, of whom six were children. In thirty-three of these there were plain clinical indications of heart disease. The remaining patients, though free from cardiac affections, were suffering from serious diseases. Seven were the subjects of severe emphysema, while two were afflicted with rheumatic fever and exophthalmic goitre concurrently. The therapeutic measures necessary for the treatment of the group of fifty-five patients covered a wide range, and included the use of iodine, protein shock therapy, salicylates, injections of a vaccine of *Bacillus typhosus*, and the intramuscular exhibition of a preparation of bismuth. None of this treatment was modified or discontinued during the administration of chloral hydrate. The latter drug was employed in the official dose of twenty grains. Two doses, each of ten grains, were given during the day and one dose of twenty grains at night. In these amounts the drug produced sleep in about thirty minutes, and the sleep continued as a rule until the awakening of the patients seven or eight hours later. Continued for a week or ten days, the average amount of chloral used was about 350 grains for each patient.

Alstead confidently asserts that chloral hydrate is an effective hypnotic, leading neither to tolerance nor to habit formation. In frequent doses it may produce redness of the eyelids and lachrymation, and is best avoided, except when greatly diluted, in cases of gastric and intestinal disorder. Apart from these qualifications, however, there seems no contraindication to the use of the drug. Not only is there no evidence available to prove any weakening of cardiac action after chloral hydrate, but there were actually signs of clinical improvement after its use in two cases of Alstead's series. In no instance did the electrocardiograms show any changes which might be taken as indicating depression of conductivity resulting from the hypnotic.

Although the blood pressure fell in 50% of the patients, the fall never exceeded 15 millimetres of mercury and was only such as might have been expected to follow continued rest and a long sleep. Strangely, about one-quarter of Alstead's group of patients showed an actual rise in the readings, a phenomenon no doubt due to an improvement in general condition. These observations, if further corroborated, may lead to the wider use of a hypnotic unlikely to lead to addiction, if only by reason of its unpleasant taste.

[1] *The Lancet*, April 25, 1936.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Radiation of Heat from the Human Body: The Transmission of Infra-Red Radiation through the Skin.

JAMES D. HARDY AND CARL MUSCHENHEIM (*Journal of Clinical Investigation*, January, 1936) report further results on the emission, reflection and transmission of infra-red radiation by the human skin. Their observations were made by means of a rock-salt prism spectrometer designed and built in the laboratory of the Russell Sage Institute of Pathology in affiliation with the New York Hospital, New York. Since nearly all observers agree that it is only within the near infra-red range that any appreciable penetration of the skin occurs, it is this portion of the spectrum, if any, which is effective in the deep penetration of radiant heat, and it is this portion of the spectrum which these workers have investigated. Since infra-red radiation, as at present used, penetrates in effective amounts, it was thought desirable to repeat the observations on living skin as well as on dead skin. Observations both of direct transmission and scattering were made on: (i) relatively thick specimens of dead human skin obtained from surgical amputations, (ii) the entire thickness of a rabbit's ear, first alive and later amputated, and (iii) thin epidermal pieces of human skin obtained by cantharides blister from the volar surfaces of the forearm of living subjects. As it was found in preliminary experiments that the method of sponging the dead specimens with saline solution to prevent excessive drying was not entirely effective in preventing drying and consequent change in penetrability of the tissue, this method was abandoned, and the entire specimen, with aperture frame, was kept immersed in the normal saline solution between readings. These authors' work shows that infra-red transmission through the skin, even of the most penetrating rays, is of small proportion, about 95% of these being absorbed within two millimetres of the surface and 99% within three millimetres of the surface. The authors conclude that the heating effect of infra-red radiation is exerted principally on the body surface and that whatever therapeutic effect is obtained by such radiation is due to this local effect and not to deep penetration of the rays, which is negligible in amount. Photography of the human body in infra-red light, while yielding detail not obtainable by ordinary photography, will not yield pictures of structures more than a few millimetres under the surface and, owing to scattering, will not give sharp detail. The absorption spectrum of normally wet skin is essentially that of liquid water. Upon drying, other absorption bands not due to water become evident.

Fœtal Circulation and Respiration.

J. BARCROFT (*Physiological Reviews*, January, 1936), in a review of experimental work on fœtal circulation and respiration, points out that different mammals have different periods of gestation and are born in very unequal degrees of development. These facts have constantly to be borne in mind when observations made upon the physiology of the embryo in one form are compared with those made in another form. For example, the sheep, though it has a longer period of gestation than the dog and a shorter period than man, is born in a much more developed condition than either. A number of observations have been made of the fœtal blood volume, the arterial and venous pressure in the fœtus, the pulse rate, the vascular reflexes, and the exchange of gases between fœtal and maternal blood. But there is no animal for which all this information can be given, and the author warns his readers against the application of information obtained upon one animal to patch up discrepancies in that obtained from another. It is not clear that the mechanism of placental respiration is the same in all mammals; indeed it seems clear that it is not so. His object is to put before the reader such few facts as are known about the circulation and respiration in the sheep and the goat, with notes on other animals by way of comparison. The data required for a complete exposition of the exchange of gases in the placenta are the following: (i) the rates of blood flow in the fœtal and maternal circulations; (ii) the percentage saturations with oxygen and the carbon dioxide contents of the blood reaching and leaving the placenta; (iii) the oxygen and carbon dioxide dissociation curves. All are agreed that the blood of the fœtus has a greater affinity than that of the mother for oxygen. About the cause of the difference, opinions expressed are more at variance. Most workers agree that the curve of the mother is displaced as compared with the normal and has a less affinity for oxygen than the normal. There is good reason for attributing this change to an alteration of the hydrogen ion concentration in the blood. Where there seems to be less unity is with regard to the fœtal curve. The divergences of the fœtal curve from normal and the fact that these could not be explained on the ground of abnormal alkalinity suggested an investigation of the properties of fœtal hæmoglobin. In no form of life at present studied have the fœtal and maternal hæmoglobins been identical. Naturally, great interest attaches to the transition from the fœtal to the maternal hæmoglobin which takes place at birth. This subject, however, is outside the scope of the author's article.

Rickets and Scurvy on Adequate Diets.

ALAN MONCRIEFF (*Journal of Physiology*, December, 1935) reports two cases which illustrate difficulties in the simple ætiology assumed for the deficiency diseases. The two children concerned were recently under his care at the Hospital for Sick Children, Great Ormond Street. The first case was that of a female child of seventeen months, artificially fed from birth on a rational diet. Until one year of age the child received in addition one drachm daily of a good cod liver oil, then a preparation containing cod liver oil and Parish's food in equal parts; half an ounce of this preparation had been given daily. For two months previous to her admission to hospital she had received another preparation reputed to be twenty-five times as rich as cod liver oil in vitamin *D*, and of this she had ninety minims daily. Admission to hospital was sought for a "fit" and general irritability dating from bronchitis six months previously. Clinically the child presented all the signs of rickets and there was also evidence of tetany in the form of facial irritability and *laryngismus stridulus*. Her serum calcium was 4·3 milligrammes per 100 cubic centimetres (ionic calcium 1·9 milligrammes) and the inorganic blood phosphorus 3·2 milligrammes per 100 cubic centimetres. X ray examination revealed florid rickets. There was no evidence of renal disease or of cœliac disease. The author states that improvement has been slow with adequate vitamin *D* intake and calcium given intravenously. The second case was that of a male child, aged ten months, who had been breast-fed for some weeks and who was then changed to a boiled milk and water mixture with some attempt at mixed feeding for a few weeks. He had been given orange juice since birth, amounting to half an orange daily for many months, and had been perfectly well in every way until three days before his admission to hospital, when he appeared to have pain in his legs. On admission he had the classical tenderness in the legs of scurvy, typical changes in the gums round two erupted teeth, hæmaturia (microscopic) and X ray changes at the ends of most of the long bones. In view of the apparent non-absorption of vitamin *C* he was given 400 milligrammes of ascorbic acid intravenously. By next day the child was less miserable, and within forty-eight hours there was obviously less tenderness. The child has now been accustomed to a good mixed diet with fruit juice daily, and the X rays show definite healing changes.

BIOLOGICAL CHEMISTRY.

The Male Hormones.

THE method of assay of "Testosterone" and its effects on castrated and ovariectomized rats have been described by V. Korenchevsky, M. Dennison and I. Brovsin (*Biochemical Journal*, March, 1936). In the castrated males injections of "Testosterone" restored to normal the weights of the adrenals, thyreoids, liver, kidneys and heart, and increased the rate of involution of the thymus; the body weight was increased to or even above the normal without definite changes occurring in the deposition of fat. Although the sexual organs (seminal vesicles, prostate, penis) were increased in size, the doses used did restore these organs to normal weight. Injections of "Œstradiol" effected changes similar to those obtained after "Œstrone", that is, considerable increase in weight of the seminal vesicles and smaller increases in weight of prostate and preputial glands. "Œstrone" and "Œstradiol" injected with "Testosterone" showed only slight cooperative effect on the weights of the seminal vesicles and prostate. In ovariectomized females, "Testosterone" injected alone produced hypertrophy of the atrophied sexual glands, but not up to the normal weight. The weight of the thymus was reduced. There was a definite cooperative effect between "Testosterone" and "Œstrone" in restoring the sexual glands to or towards the normal weight and in increasing the rate of involution of the thymus. It was shown that the male hormones had some of the important properties of the female hormones. A comparison was made of the biological properties of "Testosterone", "Androsterone", "Androsterone-diol" and the gonadotropic hormone.

The Anti-Pernicious Anæmia Principle in the Stomach.

As the result of experiments on the treatment of pernicious anæmia, E. A. Greenspon (*The Journal of the American Medical Association*, January, 1936) concludes that it is unnecessary to postulate the "extrinsic factor" of Castle in a theory to explain the function of the stomach in relation to the causation of pernicious anæmia. "Ventriculin" (a desiccated, defatted hog stomach preparation) was incubated with pepsin and hydrochloric acid and then fed to a relapsing pernicious anæmia patient. No reticulocyte response occurred. The same patient was then fed the untreated "Ventriculin". After six days the reticulocytes reached a value of 29·3%. Pepsin was removed from fresh frozen gastric mucosa by acetone extraction. The depepsinized preparation, in doses of 40 grammes per day, gave a good reticulocyte response and continued treatment produced a satisfactory remission. Treatment of the depepsinized gastric mucosa with pepsin and hydrochloric acid rendered it inactive; treatment with hydrochloric acid alone did not destroy its activity. Two normal subjects, after oral administration of calcium carbonate to neutralize the gastric hydrochloric acid, were given injections of histamine to stimulate the secretion of gastric juice, which was removed by means of a Rehfuss tube, collected in an ice-cold vessel and immediately neutralized. A pernicious anæmia patient was given 250 cubic centimetres of this gastric juice each morning on an empty stomach. No food was allowed for a period of four hours after the dose was taken. A typical reticulocyte response occurred on the seventh day of treatment. Pepsin is therefore considered to be antagonistic toward the gastric anti-pernicious anæmia factor.

Estimation of Alcohol.

H. NEWMAN (*Journal of Pharmacology and Experimental Therapeutics*, March, 1936) has described a method for the estimation of alcohol in body fluids. The method is sufficiently simple and rapid for the clinical estimation of alcohol in blood in connexion with alcoholic intoxication. The fluid to be analysed is placed over anhydrous sodium sulphate, the alcohol is removed by distillation under reduced pressure at 40° to 45° C. and is collected in an oxidizing solution (N/10 $K_2Cr_2O_7$ in equal volume of concentrated H_2SO_4). The alcohol is oxidized by the dichromate, which is back-titrated with N/10 sodium thiosulphate. In tests in which alcohol-water and alcohol-blood mixtures were used, 96% to 99% of the alcohol was recovered.

Iron in Human Nutrition.

AN investigation of the iron intake of 63 men and 63 women of English middle class, living on freely chosen diets, has been made by E. M. Widdowson and R. A. McCance (*Journal of Hygiene*, February, 1936). The daily intakes of the men averaged 16·8 milligrammes of iron; of the women, 11·4 milligrammes. The "available" iron intakes averaged 10·8 milligrammes for men (64% of total iron) and 7·9 milligrammes for women (76% of total iron). The higher percentage availability of the iron in the women's diets was due to the fact that a smaller proportion of the total iron was derived from meat (average daily meat intake for men, 5·2 ounces; for women, 3·1 ounces). Hæmoglobin estimations were made on blood samples from 50% of the subjects; values for the men averaged 102% (100% equivalent to an oxygen capacity of 18·5 volumes of oxygen per 100 cubic centimetres) and for the women 93%. Although no correlation was observed between the total or available iron intake and the hæmoglobin level, it was found that large doses of iron (100 milligrammes as ferrous sulphate or ferric ammonium citrate) increased the hæmoglobin level in women by 4% to 17% (average 10%), while in the men the level was not significantly changed.

Sodium Salts and Carbohydrate Metabolism.

I. MCQUARRIE, W. H. THOMPSON AND J. A. ANDERSON (*Journal of Nutrition*, January, 1936) have studied the effects of ingestion of excessive quantities of sodium and potassium salts on the carbohydrate metabolism and the blood pressure in one normal and four diabetic children. One diabetic child required a daily intake of 60 to 90 grammes of sodium chloride to satisfy his craving for salt. It was concluded that the ingestion of one to two grammes of sodium chloride per kilogram of body weight daily exerted a favourable influence on the carbohydrate metabolism when the diabetic children were maintained on a simplified diet low in potassium. There was a definite reduction of the glycosuria, together with a rise of systolic and diastolic pressures. The high blood pressure levels were maintained as long as the salt was taken. The shape of the sugar tolerance curves was not influenced by the excessive salt ingestion. In severe diabetes ketonuria appeared earlier after the withdrawal of insulin when the sodium chloride intake was low than when it was very high. Potassium chloride showed the reverse effect on both glycosuria and blood pressures; when given in doses of 10 to 20 grammes per day with a simplified diet low in sodium, there was a slight fall in systolic and diastolic blood pressures and a significant increase in the degree of glycosuria. When the sodium and potassium salts were administered simultaneously, one part of potassium was found to abolish the effects of three chemically equivalent parts of sodium.

Vitamin G and Canine Black Tongue.

C. J. KOEHN, JUNIOR, AND C. A. ELVEHJEM (*Journal of Nutrition*, January, 1936) have shown that canine black tongue, which is considered to be comparable with human pellagra in symptoms and ætiology, can be cured by administration of vitamin *G*. Typical black tongue symptoms were produced in dogs by feeding them with a modified Goldberger ration. Hepatoflavine, prepared from liver extracts by adsorption on fuller's earth, was completely inactive in the prevention or cure of black tongue. Fractions, prepared from liver extract, rich in vitamin *G* (B_2) and from which the hepatoflavine had been removed, were very active in the cure of black tongue.

British Medical Association News.

SCIENTIFIC.

A MEETING of the Victorian Branch of the British Medical Association was held at the Royal Melbourne Hospital on May 20, 1936. The meeting took the form of a series of clinical demonstrations.

Peptic Ulceration.

DR. W. A. HAILES showed a man, aged forty years, who for twenty years had suffered from pain in the epigastrium which came on about one hour after meals, the attacks occurring every four or five months. Five years earlier the attacks had become more severe, of longer duration and more frequent. Two months before his admission to hospital in February, 1936, the patient had a hæmatemesis, after which pain was relieved only slightly by alkaline powders, vomiting or food. Six days before his admission to hospital he had had a further hæmatemesis, with almost continuous pain until admission. The barium meal examination showed the presence of an ulcer high up on the lesser curvature. Dr. Hailes had thought that it would be difficult to exclude a neoplasm. The test meal revealed the absence of free hydrochloric acid, and a skiagram taken twelve months earlier had shown no evidence of peptic ulcer. A large ulcer, 6·25 by 3·75 centimetres (two and one-half by one and one-half inches), which was adherent to the pancreas high up on the lesser curvature was removed at an operation performed on March 3, 1936, but gastro-jejunostomy was not done because of the poor condition of the patient. After a transfusion his condition gradually improved, although he could only take small quantities of food at a time. When a second barium meal examination was carried out four weeks after the operation, definite delay in emptying of the stomach and a large residue below the pylorus at the end of forty-eight hours were revealed. Six weeks after the first operation gastro-jejunostomy was performed successfully, and the patient was able to take food comfortably.

False Aneurysm of Axillary Artery.

Dr. Hailes also showed a man, aged twenty-one years, who two and a half weeks before admission to hospital had stumbled over a log and shot himself in the chest with a pea-rifle Complete paralysis of the arm set in immediately, and pain commenced down the ulnar border of the forearm and hand on the day following the accident, and had persisted. On examination of the wound the entry of the bullet was found to be 2·5 centimetres (one inch) above the lower border of the *pectoralis major* muscle and about 2·5 centimetres from the anterior axillary fold. The patient's wrist and finger movements were absent, the supination of the forearm was weak and pronation absent; all the shoulder movements were present but weak, and flexion of the elbow was weak and extension absent.

Sensation was absent in the hand on the ulnar side of the dorsum and over the volar surface, and diminished on the radial side of the dorsum, but prolonged stimulation produced a burning sensation; the patient had a burning sensation in the forearm on stimulation of the dorsum, with anæsthesia along the ulnar border, but sensation was normal on the volar surface. The blood pressure was equal in both arms and sensation above the elbow was normal, with strong expansile pulsation detectable over a considerable area in the axilla. An operation was performed eight days after the patient's admission to hospital, and a false aneurysm of the third part of the axillary artery commencing at the level of the junction of the two roots of the median nerve was found. The artery was ligated above and below the aneurysm and a subcapsular branch was ligated; all the nerves were found to be intact; the loss of blood during the operation was considerable, and a transfusion was given. After the operation there was no pulse palpable in the arm and the hand was cold, but it regained warmth after twenty-four hours. Sensation had only improved slightly, and was normal in the dorsum of the forearm, but no muscle power had returned eighteen days after the operation. For a week after the operation the pain persisted, but since then had been practically absent.

Fracture-Dislocation of the Talus.

Dr. Hailes also showed a man, aged thirty-nine years. Seven weeks earlier the patient had fallen from a scaffolding about eight feet from the ground onto the outer side of his foot, so that it was sharply inverted. A skiagram showed the talus to be laterally dislocated on the *os calcis* and dislocated from the scaphoid, so that the head of the talus projected on the lateral surface of the foot, and there was also a fracture of the body of the talus with displacement. Reduction, after being unsuccessfully attempted by manipulation, was then attempted by open operation, but the talus could not be kept in position owing to gross comminution of the bone. The talus was removed, and the foot was placed in plaster with the heel displaced backwards as far as possible and the foot somewhat inverted. The foot had remained in plaster for five weeks, but the patient had been walking on it for the past week.

Compound Supracondylar Fracture of the Humerus.

Dr. Hailes's next patient was a male, aged twenty-three years. The patient received a glancing blow on his elbow in a flexed position on March 26, 1936. A compound fracture of the supracondylar portion of the humerus with anterior displacement of the distal fragment resulted. Twelve days after the accident a Kirschner wire was inserted through the olecranon process of the ulna, and traction was exerted on the humerus by means of a Böhler traction apparatus. The arm was then placed in a complete plaster with the elbow extended, a window being left over the original wound; the fragments were all in good position. After fourteen days the plaster was removed and the elbow flexed gradually to 90°; then a second complete plaster was applied, and three weeks later that plaster was removed and further skiagrams were taken.

Fracture-Dislocation of the Wrist.

Dr. Hailes's last patient was a woman, forty-five years of age. Thirteen weeks before admission the patient had fallen down a stairway, fracturing the ulnar styloid and distal end of the radius of the right hand; there was also a lateral subluxation of the carpal bones, except the semilunar, which was dislocated forwards but not laterally. Kirschner wires were inserted through the olecranon of the ulna and through the bases of the metacarpal bones, and traction was applied by means of a Böhler extension apparatus; the dislocation was completely reduced and the fracture was placed in good position. A complete plaster was then applied from the heads of the metacarpals to the elbow joint, with the wrist joint only slightly extended. Five weeks later, when the plaster was removed, movements of the wrist joint commenced.

A Clinico-Pathological Demonstration of Intracranial Aneurysms.

DR. E. GRAEME ROBERTSON referred briefly to the records of some cases at the Royal Melbourne Hospital, and gave a brief account of the dramatic symptoms produced by rupture of the intracranial aneurysm in these patients—headache, vomiting and unconsciousness, followed by signs of cerebral compressing and meningeal irritation, and, in fourteen out of twenty-six cases, death. He indicated the site of the aneurysm in those cases which came to autopsy. To illustrate this he showed several specimens, including one in which blood had spread along the pia-arachnoid sheath of the optic nerve, producing a large subhyaloid hæmorrhage. He then referred to recent developments

in the surgical treatment of intracranial aneurysms, of which Norman Dott was the chief exponent, especially dealing with the accurate localization which cerebral angiography offered. He then showed an angiogram demonstrating an aneurysmal sac in a patient who suffered from severe headache and giddiness, following the sudden onset of pain in the back of the neck, loss of consciousness and twitching of the right arm. Next he showed an angiogram of an aneurysm of the internal carotid artery, causing the cavernous sinus syndrome of paralysis of ocular muscles, dysæsthesiæ and reduced sensation over the distribution of the ophthalmic and maxillary divisions of the fifth cranial nerve and optic atrophy.

Dr. Robertson then demonstrated an aneurysm of the middle cerebral artery which was embedded in the uncus, producing headaches and uncinate epilepsy, an acid smell increasing with each breath and merging into a feeling of dizziness with a "familiarity" psychological content.

Next Dr. Robertson referred to calcified rings seen in skiagram films of the skull, showing several examples. Trauma was the possible cause of an aneurysm of the internal carotid artery seen in one of the films, the clinical picture being a partial third nerve palsy and extrapyramidal rigidity.

Dr. Robertson demonstrated by microscopic preparations the structure of the wall of the aneurysmal sacs and the congenital defect in the muscularis which predisposed to aneurysmal dilatation, as was first suggested by Wiley Forbes.

Leaving the subject of congenital aneurysms, Dr. Robertson showed upon an arteriosclerotic middle cerebral artery an aneurysm which had ruptured, causing sudden unconsciousness and death. Lastly, he demonstrated specimens from a young patient who gave an early history of migraine, and, since 1932, of sensory epilepsy. During the last year painful swellings had appeared and disappeared on his extremities, and finally, after a subarachnoid hæmorrhage, he died from the intracerebral rupture of a mycotic aneurysm. During life the cardiac sounds were normal and no symptoms of cardiac embarrassment were present. Small vegetations of bacterial endocarditis were seen on the mitral valve, which showed evidence of previous rheumatic disease. Microscopic preparations gave evidence of inflammatory changes in the wall of the aneurysm, contrasting with the fibrous wall of the congenital aneurysm.

Demonstrations of Skiagrams.

DR. HOWARD F. PRAAGST showed a series of skiagrams, including examples of: neurotrophic arthropathy affecting the knee and ankle in tabetic patients, and the shoulder in a case of syringomyelia; multiple lung abscesses following a peritonsillar abscess; bronchogenic carcinoma demonstrated by lipiodol injection; multiple congenital cysts of the lung; and multiple aneurysms of the aortic arch.

Of particular interest were the films of a man, sixty-six years of age, who gave a history of dysphagia for four months; the films showed the upper half of the œsophagus to be greatly dilated and almost completely obstructed by multiple pedunculated growths of varying sizes. Microscopic section of several of the smallest of these removed endoscopically showed them to be lipomata. Dr. Praagst pointed out that benign tumours of the œsophagus were very rare, and, of those that occurred, the majority were lipomatous or myomatous in structure.

Dr. Praagst also demonstrated a case of coarctation of the aorta in a man, thirty-two years of age, which illustrated clearly the characteristic smooth erosion of the lower border of the ribs, due to the hypertrophy of the intercostal vessels taking part in the collateral circulation.

Of neurological interest were the films shown by Dr. Praagst of two cases of *osteitis deformans* in which the involvement of the spine had led to compression myelitis in the dorsal region as a result of the softening and collapse of the involved vertebral bodies.

Disorders of the Blood.

DR. KEITH D. FAIRLEY presented four cases illustrating various disorders of the blood.

The first patient, twenty-four years of age, had been shown at the corresponding meeting a year ago, when he was recovering from aplastic anæmia due to benzol poisoning. By that time the hæmoglobin had risen from 19% to 67%, the red cells from 1,000,000 to 3,000,000, and the leucocytes from 1,000 to 2,600 per cubic millimetre. On May 4, 1936, the hæmoglobin was 94%, the red cells numbered 4,700,000 and the leucocytes 5,700 per cubic millimetre. The platelets, which originally numbered 43,000, had then risen to 176,000 per cubic millimetre. The patient was presented to show the relatively slow regeneration of the platelets. The use of "Nucleoproteid K 96", to hasten regeneration of the leucocytes, had been considered, but since it was determined that there was a gradual increase in the proportion of leucocytes, this had not been used.

The second patient shown by Dr. Fairley was a man, thirty-four years of age, who had been treated for pernicious anæmia for four years before coming to hospital. During that period he had also suffered from *spondylitis ankylopoietica*. There had been three exacerbations of the anæmia, with good recovery from the first and a fair, though brief, recovery from the second. The present exacerbation commenced in December, 1934, and the patient had been confined to bed for the last two months, during which time frequent injections of "Campolon" had failed to effect any improvement. Though no secretion of hydrochloric acid followed the ordinary test meal, some appeared in response to histamine. The Van den Bergh test resulted in a delayed reaction, four units of bilirubin being present. The hæmoglobin was 33% (4·7 grammes), the red cells numbered 1,870,000 and the leucocytes 8,100 per cubic millimetre. The halometer reading gave an average diameter for the red cells of 7·3. The reticulocytes numbered 22%, and a high reticulocyte count persisted. On November 9, 1935, the hæmoglobin was 34% and the reticulocytes were 15·6%. The fragility of the red cells to hypotonic saline solution was normal. The serum failed to react with the Wassermann test. Apart from the spondylitis and evidence of some arthritic changes in both knee joints, a lemon-yellow tint of the skin was the only abnormal clinical finding. The temperature ranged from 36·7° to 37·8° C. (98° to 100° F.) daily before the splenectomy, but a month later the fever had subsided, and the temperature did not exceed 37·2° C. (99° F.) on any occasion during the remaining ten weeks of his stay in hospital.

A diagnosis of chronic hæmolytic anæmia of unknown origin was made on account of the persistent reticulocytosis and the increased bilirubinæmia. Transfusions on November 8 and 18, each of 300 cubic centimetres (ten ounces) of blood, raised the hæmoglobin to 54% and the red cells to 3,400,000 per cubic millimetre, while the reticulocytes fell to 4·6% before splenectomy was performed on November 29, 1935. The spleen was much enlarged and congested, but did not show any gross fibrosis. Considerable oozing occurred from numerous adhesions on the diaphragmatic surface. By December 2 the hæmoglobin had fallen to 37%, the red cells to 2,400,000 per cubic millimetre, while the reticulocytes rose to 9·4%. Thereafter the hæmoglobin and red cells increased steadily until on May 12, 1936, the findings were 67% and 4,500,000 per cubic millimetre respectively. The leucocytes numbered 8,500 per cubic millimetre, the reticulocytes 1·4% and the average size of the red cells was the same as before the operation, namely 7·3. Convalescence was interrupted by the supervention soon after the operation of a left-sided pleural effusion, which subsided after two aspirations. Subsequently the clinical condition improved, corresponding with that of the blood.

Dr. Fairley said that this patient had shown considerable improvement after the two transfusions of blood, but the excellent result from splenectomy in a similar case suggested its use in this case, although the spleen was not palpable. The result to date was fairly satisfactory.

Large doses of iron, which had been taken since before the operation, were being continued.

Dr. Fairley showed another patient, a woman, thirty-eight years of age, who was first seen on June 27, 1930, with a history of anæmia for the preceding five years. Her father was said to have died at the age of fifty-four years from Banti's disease. The patient was pale, with a slight lemon-yellow tint. A hard tender spleen extended 5·0 centimetres (two inches) below the ribs, and the liver edge was palpable one finger's breadth below the costal margin. A delayed response to the Van den Bergh test was obtained on two occasions, 2·5 and 3·5 units of bilirubin being present. The fragility of the red cells to hypotonic saline solution was normal. The serum had failed to react to the Wassermann test. The hæmoglobin value was 45%, and the red cells numbered 4,250,000 per cubic millimetre. The films revealed corresponding hypochromia of the red cells, some poikilocytosis, and occasional polychromasia and basophilic stippling. Platelets were numerous, and there was a moderate relative lymphocytosis and the reticulocytes were not increased. These features had been present in practically all blood films subsequently examined, and in addition anisocytosis was a prominent feature, many microcytes and some macrocytes being present. In view of the fact that the diagnosis in the case of her father might have been incorrect and because of the splenomegaly with increased bilirubinæmia, splenectomy was performed on September 18, 1930. The liver was more grey in colour than usual, and histological examination of the spleen revealed some arteriosclerosis and slight fibrosis. On August 26, 1931, a total hysterectomy was performed for chronic endometritis and an ovarian cyst. For the next few years the patient attended only at long intervals and refused to take iron with any regularity. Her subsequent progress was fairly typical of idiopathic hypochromic anæmia. Repeated examinations revealed hæmoglobin values from 40% to 50%, with red cell counts about 5,000,000 per cubic millimetre. The red cells, however, showed a definite tendency to macrocytosis with some microcytes, halometer readings giving an average red cell diameter of from 7·6 to 8·0. Obvious hypochromia was constantly present. This tendency to macrocytosis, as judged by the halometer readings, in association with a hypochromic anæmia, had been noted by Dr. Hilda Gardner in several patients after a splenectomy performed for various blood disorders, and these cases were being investigated in more detail. In November, 1933, dysphagia became troublesome, and œsophagoscopy revealed some spasm of the crico-pharyngeal muscles, while no actual obstruction was seen on radiological examination. Glossitis was found to be present, and the nails were thin and rather flat. The condition corresponded with the Plummer-Vinson syndrome, and temporary, incomplete relief resulted from dilatation of this area of spasm. On April 2, 1936, the patient was admitted to hospital for intensive iron therapy; iron and ammonium citrate, in a dose of four grammes (sixty grains), thrice daily, raised the hæmoglobin value from 51% to 69% in sixteen days with a corresponding clinical improvement, and since the patient's discharge from hospital that therapy had been continued with further satisfactory results.

Dr. Fairley commented that the condition was apparently one of idiopathic hypochromic anæmia, and it was doubtful whether splenectomy was necessary, though a typical case of this condition did not show an increased bilirubinæmia.

Paroxysmal Hæmoglobinuria a Frigore.

The last patient shown by Dr. Fairley was a man, sixty-two years of age, who was first seen by him on July 3, 1935, when he had stated that on exposure he suffered from a dull ache in the loins, extending to the groins and testes, for a few days after which the urine had the dark colour of blood, and the tongue became blue; but there appeared to be no urticarial manifestations. The first attack had occurred at the time of the flood in December, 1934. Forty years earlier he had contracted gonorrhœa. On examination, the liver and spleen were seen to be moderately enlarged, and mild signs of hypothyreoidism were noted, but no hæmoglobin was detected in the urine; the last attack had occurred a few days previous to the meeting. The serum fixed fifty-four units of complement in the Wassermann test, with the technique of York and McFie. (The patient's serum was added to his own or to other normal red corpuscles and was left for seven minutes at 0° C., and then for thirty minutes at 37° C.; appropriate controls with normal serum were used.) It was shown that a hæmolysin capable of causing autohæmolysis after exposure to low temperatures was present in the patient's serum. In the attack this hæmolysin united with the red cells when they were cooled during their passage through the skin vessels, and hæmolysis occurred when complement came into action in the warmer parts of the body. Since the commencement of treatment, first with mercury and iodide by mouth, and later by bismuth injections, no attacks had occurred, possibly because of the greater care with regard to exposure to cold, and the serum now fixed twenty units of complement in the Wassermann test. The case was a classical example of paroxysmal hæmoglobinuria a frigore.

Lupus Erythematosus.

DR. JOHN H. KELLY showed a woman, forty-two years of age, who was suffering from extensive *lupus erythematosus*. A small residuum showed the nature and extent of the lesions which had occupied the nose, the bat's wing area of the face, scalp, manubrium of the sternum, as well as the back of the hands. The result had been obtained by the intravenous use of gold sodium thiosulphate and the intramuscular use of "Neo-Cardyl". It was pointed out that not all cases of *lupus erythematosus* responded so well to gold therapy, and that in such cases certain additional factors would be found; these would include septic focal conditions and any factors disturbing the vasomotor mechanism, such as worry and anxiety. *Lupus erythematosus* would be better described by the term *erythema centrifugum*, as it was a fixed erythema of the skin, with sharp demarcation, showing central atrophy and exhibiting spread by extension of its peripheral border. *Lupus erythematosus* was not generally regarded as a manifestation of tuberculosis. This condition was contrasted with *lupus vulgaris*.

Lupus Vulgaris.

The next patient shown by Dr. Kelly exhibited *lupus vulgaris* of the left cheek. There was a central area with atrophy and typical apple-jelly nodules at the periphery. Dr. Kelly stated that *lupus vulgaris* was somewhat commoner in Victoria than was generally believed, and that he had in recent years seen three children inoculated from cases of open pulmonary tuberculosis. In this particular patient under review diabetes had rendered an already difficult condition very recalcitrant to treatment. The individual apple-jelly nodules consisted of giant cell systems in which tubercle bacilli were actually present, and treatment aimed at producing necrosis of these nodules by the agency of ultra-violet light in the form of the water-cooled Kromayer lamp (a substitute for Finsen light), or by such chemicals as acid nitrate of mercury. Old tuberculin injections were also given. *Carcinoma in lupo* was a possibility in all old cases of *lupus vulgaris*, and this tendency was probably accelerated when radium was used as a form of therapy. Radiotherapy should have no part in the treatment of *lupus erythematosus* or *lupus vulgaris*.

Multiple Rodent Ulcers.

Dr. Kelly's next patient was a man, forty-eight years of age, exhibiting three large, more or less rounded, easily bleeding ulcers of the forehead; these lesions had been extending for five years without effective treatment. Examination showed that they had the characteristic pearly edge that was associated with basal-celled epithelioma. In this particular patient the Wassermann

test gave a negative result, histological section showed basal epithelioma, and radium therapy was to be applied later. Dr. Kelly said that such multiple superficial rodent ulcers were uncommon and that this type did not seem to be associated with the rodent ulcers coexisting with solar keratosis. In this case it was pointed out that the lesions occurred on the forehead, an area fairly well protected from the action of the sunlight by a hat.

Multiple Epidermophytosis.

Dr. Kelly also showed a man, twenty-eight years of age, who had several annular lesions on the legs, arms and body. Dr. Kelly pointed out that the groins were affected in the fashion characteristic of Dhobie's itch or *epidermophyton inguinale*. The border of the lesions, wherever examined, showed erythema and undermined epidermis with a more or less ragged scale, and in the groins there was a tendency to festooning, though there was no abnormality of the skin between the toes. Differential diagnosis between ordinary intertrigo of the groins and Dhobie's itch lay in the sharply defined scaly border and rather quiescent central area of Dhobie's itch explaining the old name of *eczema marginatum*, compared with the uniformly red, scaly, and perhaps moist and raw condition found in common intertrigo. It was pointed out that it was frequently necessary to make a microscopic examination to detect the presence of the fungus, which could be found in the form of long, branching, segmented filaments.

Dr. Helen Gardner, of the Walter and Eliza Hall Institute, had set up a microscopic specimen demonstrating the fungus present. One of the points of interest in this case was that the patient was employed in a rubber company and believed his condition to be occupational in origin; inquiry, however, showed that he was in the habit of working on a yacht in the week-ends and wearing old bathing trunks, which were left on board. Treatment consisted in the use of a suitable fungicide, such as weak Whitfield's ointment (3% salicylic and benzoic acid in a suitable base).

Red Leg.

The last patient shown by Dr. Kelly was a woman, forty-two years of age, with general œdema, redness and exfoliation of the left leg. The damaged area extended from a uniformly well defined upper margin at the knee level to the dorsum of the foot, where there was a clearly distinguishable lower edge just proximal to the plantar surface, which was perfectly normal. Dr. Kelly demonstrated that this condition was frequently unilateral, and when bilateral was often worse in one limb than in the other. It was sometimes confused with varicose dermatitis, but whether varicose veins were present or not, this condition, termed red leg, was a clear and well defined entity. In some of the text-books it was described as gravitational eczema, but a careful history of each case showed that its true nature was that of friction dermatitis. The patient's hands and feet produced the damage by vigorous rubbing which was carried out at night. This condition was usually preceded by some trauma, such as a Pott's fracture, old burn scar, or an operation for varicose veins. Red leg, or friction dermatitis, of the lower extremity, with the features given above, was fundamentally a pruritus, having its origin in psychological factors with resulting physical manifestations produced by the patient's own activity. The psychological problems confronting this particular patient were discussed; treatment consisted in eliciting and explaining the facts to the patient, then admitting her to hospital and applying appropriate remedies to the affected area. Dr. Kelly said that the limb should be immobilized and protected by the use of a back splint, with side Clines splints and plentiful bandaging or similar restraints. The patient should also be helped by ascertaining the directions in which the psychological difficulties could be overcome. This might involve advice regarding such factors as environment, mental and physical diversions and conditions of employment. It would be only after such a comprehensive psychological survey that one could hope to cure these patients. Though he indicated that the general physical health should receive adequate attention, Dr. Kelly did not go into details. In conclusion he pointed out that Unna paste stockings, "Elastoplast" and other coverings were quite useless from the standpoint of protective therapy in the condition described as red leg.

Hodgkin's Disease.

DR. K. HADLEY showed a patient, twelve years of age, who had been referred from the Children's Hospital in July, 1929, as suffering from Hodgkin's disease. Enlargement of the axillary and cervical glands was present and the diagnosis was established by microscopic sections. The patient was given radiotherapy in July, October and November, 1929, and at intervals throughout 1930, 1931 and 1933. In May, 1934, the patient was well, with a few small, hard, inactive glands in the right axilla and right supraclavicular region. In January, 1935, the spleen was found to be enlarged four fingers' breadth. Deep radiotherapy to the spleen had resulted in considerable diminution in its size; the patient's condition was otherwise satisfactory.

Dr. Hadley also showed a patient, twenty-two years of age, who had reported in August, 1928, complaining of a swelling in the right side of the neck, which had been present for ten weeks; it was excised and a section revealed Hodgkin's disease. The patient was treated with courses of deep therapy during 1928, 1929, 1930 and 1932; during this time there was slight glandular enlargement in the right side of the neck and in both axillæ. At the time of the meeting there were no enlarged glands palpable and the patient was well.

Dr. Hadley next showed a patient, sixty-three years of age, who had reported in December, 1933, complaining of a swelling in the left side of the neck which had been present for nine months. On examination there were huge masses of matted glands in the left side of the neck, extending from the mastoid process to the clavicle, and the spleen was palpable. A biopsy revealed Hodgkin's disease. One month after deep radiotherapy treatment in December, 1933, it was found that the masses of enlarged glands had completely disappeared, but in May, 1934, there was some recurrent glandular enlargement in each axilla, which responded rapidly to further treatment. At the time of the meeting all glandular areas were clear and the spleen was not palpable.

Another patient shown by Dr. Hadley was twenty-eight years of age, who first came under his notice in August, 1934, complaining of lumps in each axilla, the neck and the abdomen. Examination had revealed a large, tender, abdominal mass to the right of the umbilicus and enlargement of the inguinal glands of both sides, glandular masses in each axilla and numerous enlarged deep cervical glands on both sides. A biopsy revealed active Hodgkin's disease, suggesting sarcoma. All enlarged glands disappeared following the deep radiotherapy and the patient was apparently quite well.

Leuchæmia.

Dr. Hadley also showed a patient, forty-five years of age, who reported in 1933, complaining of dyspnœa, of weakness and of a lump in the abdomen. Examination showed the liver to be enlarged four fingers' breadth below the costal margin and the spleen to be enlarged to the umbilicus; the white blood corpuscles numbered 156,000 per cubic millimetre. There was a rapid diminution in the size of the spleen and fall in the leucocyte count following deep radiotherapy; the spleen, however, had recently become larger and the white count had risen to 173,000 prior to further treatment three weeks before the meeting. The patient had received radium treatment in December, 1934, for associated menorrhagia.

Dr. Hadley also showed a patient, forty-three years of age, who reported in September, 1931, with a nine-months' history of lassitude, pallor and weakness, an abdominal swelling and dyspnœa for a period of six months. Examination showed that the spleen reached to the right iliac fossa and that the white blood corpuscles numbered

705,000 per cubic millimetre. Deep radiotherapy was given and the count fell to the level of 11,000 in August, 1932. Since that time there had been gradual risings in the white count, followed by further courses of deep radiotherapy, with subsequent falls in each instance, the last course of treatment being given in April, 1936, prior to which the white blood corpuscle count had been 92,000.

The next patient to be shown by Dr. Hadley was sixty-one years of age. He had reported in April, 1932, with a history of weakness and anorexia of six weeks' duration. On examination it was seen that the spleen was enlarged one hand's breadth below the left costal margin. Blood examination showed the hæmoglobin value to be 50%; the red blood corpuscles numbered 2,600,000 and the white blood corpuscle count stood at 528,000, with numerous myelocytes. Deep radiotherapy was given in April, 1932, resulting in a rapid fall in the white count; and further deep radiotherapy was given at approximately three-monthly intervals; this succeeded in keeping the white blood corpuscle count within reasonable limits; but in May, 1936, it had risen to 71,000 and further treatment had just been completed.

Tuberculous Glands.

Another of Dr. Hadley's patients, fifty-three years of age, had reported in March, 1931, complaining of lumps in the left side of the neck which had appeared following tonsillectomy six months previously. At this time there had been a loss of two stone in weight. On examination it was found that there were enlarged glands extending from the mastoid process to the clavicle, several glands in the right axilla the size of almonds, and a somewhat larger mass in the left axilla. After deep radiotherapy had been given there was an immediate diminution in the size of the glandular masses and a sinus formed which remained unhealed for one year. At the time of the meeting the patient was well and the glandular areas had disappeared.

Dr. Hadley's next patient, seventy-two years of age, had reported in December, 1932, and complained of a lump in the right side of the neck, below the ear, which had been present for four months and which had gradually increased in size. Examination revealed that there was a hard glandular mass, four centimetres in diameter, just behind the sterno-mastoid muscle, 7·5 centimetres (three inches) below the mastoid process, and freely movable. On February 2, 1933, a mass of caseating tuberculous tissue (confirmed by section) was excised. There was a sinus extending deeply inwards towards the anterior surface of the cervical vertebræ; and the wound subsequently broke down at each extremity and enlargement of glands in the posterior triangle followed. Deep radiotherapy was administered, and in August, 1934, a small area which remained unhealed was excised, and at the time of the meeting the neck was healed and free from glandular enlargement.

(To be continued.)

NOTICE.

THE Medical Secretary of the New South Wales Branch of the British Medical Association wishes to announce that Branch meetings which are held in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, on the last Thursday of every month will begin in future at 8.30 o'clock p.m. instead of at 8.15 as heretofore.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Hughes, Geoffrey Charles, M.B., B.S., 1933 (Univ. Sydney), Sydney Hospital, Sydney.

Post-Graduate Work.

WEEK-END COURSE IN DIABETES MELLITUS AT SYDNEY.

THE New South Wales Post-Graduate Committee in Medicine announces that a week-end course in *diabetes mellitus* will be held at the Royal Prince Alfred Hospital, Sydney, on Saturday, August 15, and Sunday, August 16, 1936. The supervisor will be Dr. J. Kempson Maddox.

Programme.

Saturday, August 15, 1936, *at the Diabetic Clinic.*

2 to 2.30 p.m.—"The Present-Day Conception of Carbohydrate Metabolism", Professor C. G. Lambie.
2.30 to 3 p.m.—"Types of Diabetes, with Special Reference to Ætiology" (with clinical demonstration), Professor C. G. Lambie.
3 to 3.30 p.m.—"Biochemical Methods", Dr. E. B. Durie.
3.30 to 4 p.m.—Tea adjournment and discussion.
4 to 4.30 p.m.—"Clinical Features, Sequelæ and Prognosis of *Diabetes Mellitus*" (with demonstration of patients), Dr. I. A. Brodziak.

Sunday, August 16, 1936, *at A-2 Lecture Theatre.*

10 to 10.15 a.m.—Demonstration of instructional film.
10.15 to 11 a.m.—"Modern Dietetic Treatment" (with practical demonstration of diets), Dr. J. K. Maddox and Sister Scott.
11 a.m. to 11.15 a.m.—Morning tea.
11.15 to 11.45 a.m.—"The Use of Insulin: Reactions, Sensitivity, Hypoglycæmia", Dr. T. M. Greenaway.
11.45 a.m. to 12.15 p.m.—"Coma and Pre-Coma", Dr. Allan Walker.
12.15 to 12.45 p.m.—"Surgery and Obstetrics in Relation to *Diabetes Mellitus*", Dr. T. Farranridge.

A brief general discussion will follow.

The course will not be held unless a minimum of eight applications is received. The fee for the course is one guinea. Those wishing to attend should immediately make application to the Secretary, New South Wales Post-Graduate Committee in Medicine, the University of Sydney (telephone MW 1125).

Correspondence.

POISONING BY MIRBANE.

SIR: A case that I have in hand at present may be worth reporting in order to draw the attention of authorities concerned in the handling of "essence of mirbane" (nitrobenzol) to its very poisonous properties. My patient is a man of about fifty-five, working in certain stores. The mirbane is used in the preparation of explosives and is kept in an open tin in a shed close to where he works. In spite of the warning on the tin—"Poison: Do not inhale the fumes"—it has been very carelessly handled, spilt on the floor and on bags and allowed to stand about with the lid off, and generally treated as harmless. My patient first noticed the fumes in November last, and although they must have been fairly diluted, as he was working eight feet away from it, says that whenever he had a day of it he had headache, tiredness, weakness of legs and calves, and loss of energy, but if the wind blew the fumes away from him he felt well. Vomiting, soreness in the chest, tenderness of the tongue and roof of the mouth were also complained of.

He was exposed to the fumes daily for about sixteen weeks, and gradually got worse until he had to stay away from work. He has been away now for eight weeks and says he is gradually getting better.

His foreman, who was given a letter on the subject, written by myself, laughed at the idea, but I see that Taylor's "Medical Jurisprudence" states that "the liquid is very poisonous—twenty drops have caused death—and the vapour is much more potent than the liquid".

Several of the patient's mates who also came in contact with the fumes also complained of headache, weakness in the legs and vomiting, although their period of exposure was less than his.

I understand that measures have now been taken so that the mirbane will be handled with proper respect.

Yours, etc.,

L. P. WINTERBOTHAM.

Ipswich Road,
Annerly,
Brisbane,
Queensland,
May 16, 1936.

VITAMIN CONCENTRATES.

SIR: A child, aged five, was recently brought to my notice suffering from marked rickets, obvious clinically and radiologically. The *genu valgum* was so marked that conservative treatment was out of the question, and a senior consultant in children's surgery advised double osteotomy. The child had had no illnesses.

The interest of this case lay in the fact that the child (and the mother before its birth) had been regularly dosed with the latest vitamin concentrates. The father is a dental surgeon and well-informed about modern work in vitamins.

I was prompted to report this case by reading "Some New Drugs and Remedies", a lecture by Dr. E. W. Adams, published in *The British Medical Journal*, March 28, 1936, in which he says, speaking of vitamins: "Natural sources may have an advantage over the artificially concentrated products. Even yet we do not know all there is to be known about cod-liver oil, and recent researches suggest that there is some subtle difference between the vitamins contained in this and other fish oils, and the artificial preparations." One difference, not so subtle, is in the price.

Yours, etc.,

VICTOR J. KINSELLA.

235, Macquarie Street,
Sydney,
May 18, 1936.

CONE SHELL MOLLUSC POISONING.

SIR: Dr. Pockley's remarks about the edible nature of numerous kinds of *Conus* molluscs are very interesting.

He is surely correct when he suggests that one should speak of being "stung" by this creature rather than "bitten". No doubt it would be just as correct to speak of being "stung" by a snake, but it is not usual to speak of "snake sting"; it seems to be always "snake bite". In the *Conus* the sting is effected through the radula or ribbon tongue, in the snake by the poison fangs or teeth.

Yours, etc.,

H. FLECKER.

Cairns,
North Queensland,
May 21, 1936.

HISTIDINE TREATMENT OF PEPTIC ULCER.

SIR: I have read with interest the letter of Dr. H. G. Loughran in your issue of May 30.

Before agreeing with so important a conclusion on the subject of histidine treatment of a case of duodenal ulcer, it would be valuable to know the results of tests for occult blood, fractional gastric analysis and X ray screening for gastric duodenal motility.

The diagnosis of duodenal ulcer in this case is stated to have been made by a radiologist, and it is apparently confirmed by the effect of light diet and rest, both of which would produce similar results if the patient were suffering from hypochlorhydria and hypermotility with spasm of the first part of the duodenum. This possibility becomes the more likely following the success with "Belladonna Neutralon".

Perhaps Dr. Loughran would be good enough to provide this essential basis for the diagnosis of duodenal ulcer in this case, since his conclusions are of more than ordinary significance for us all.

Yours, etc.,

C. STANTON HICKS.

Department of Human Physiology and Pharmacology,
The University of Adelaide,
June 3, 1936.

"THE LAW OF THE SPLIT PELVIS."

SIR: I am afraid that an easy acceptance of what Dr. Gillies (this journal, June 6, page 778) terms "The Law of the Split Pelvis" may lead to some confusion in anatomical teaching. Dr. Gillies states his law as follows: "When disproportion occurs between the products of conception and the pelvic brim, separation of the ventral symphysis occurs in those types which survive." I should prefer to state it thus: When an animal supports its body weight upon its hind limbs at right angles to its body axis the ventral pelvic symphysis will be elongated. This elongation will diminish as the weight is carried on the hind limbs turned progressively into line with the long axis of the body; and the symphysis will tend to disappear altogether if the animal ceases to carry its weight on its hind limbs.

Yours, etc.,

F. WOOD JONES.

The School of Anatomy,
Melbourne University,
June 10, 1936.

University Intelligence.

THE UNIVERSITY OF SYDNEY.

A MEETING of the Senate of the University of Sydney was held on Monday, June 1, 1936.

The following degrees were conferred:

Bachelor of Medicine and Bachelor of Surgery.—Gordon David Cumming, Alan Thirlwall Denneen, Clarence Everingham, Alan Dudley Joseph Frost, Leonore Edith Jones, Arthur Geoffrey McManis, Philip Neville Simons, Jenkyn Lawrence Kenneth Williams, Arnold William Robertson.

Bachelor of Medicine.—Jack Lewis Coles, Wallace George Foulsham, Olaf McClure Spence.

Master of Surgery (Ch.M.).—Florence Charles Burke-Gaffney, M.B.

The Diploma in Public Health was awarded to Abraham Fryberg, M.B. (Melbourne), and George Muir Redshaw, M.B., B.S.

The following appointments were approved: Dr. G. O. Rudd as Lecturer in Bacteriology in the School of Medicine; Mr. Max Henry, D.S.O., B.V.Sc., as Lecturer in Epizoodemiology in the School of Veterinary Science.

On the recommendation of the Faculty of Dentistry, the Senate resolved that Latin be no longer a compulsory subject for matriculation and entrance to the Faculty of Dentistry, and that this change be put into effect at the beginning of 1937.

Obituary.

CHARLES EDWARD DOLLING.

WE regret to announce the death of Dr. Charles Edward Dolling, which occurred on June 11, 1936, at Adelaide, South Australia.

JOHN HARRY SAUNDERS.

WE regret to announce the death of Dr. John Harry Saunders, which occurred on June 15, 1936, at Potts Point, Sydney, New South Wales.

Books Received.

TEXTBOOK OF GYNÆCOLOGY, by W. Shaw, M.D., F.R.C.S., F.C.O.G.; 1936. London: J. and A. Churchill Limited. Royal 8vo, pp. 595, with four plates in colour and 234 text figures. Price: 18s. net.

DISEASES OF THE NOSE, THROAT AND EAR FOR PRACTITIONERS AND STUDENTS, edited by A. Logan Turner, M.D., LL.D., F.R.C.S.E.; Fourth Edition, revised and enlarged; 1936. Bristol: John Wright and Sons Limited. Demy 8vo, pp. 491, with 243 illustrations in the text and 21 plates. Price: 20s. net.

THE MANAGEMENT OF DELAYED LABOUR AND PROGNOSIS AND TREATMENT OF THE TOXÆMIAS OF PREGNANCY, by J. B. Banister, M.D., F.R.C.P., F.R.C.S.E.; 1936. Australia: Angus and Robertson Limited. Demy 8vo, pp. 36.

CLIO MEDICA: A SERIES OF PRIMERS ON THE HISTORY OF MEDICINE, edited by E. B. Krumbhaar, M.D.; XVI; Tuberculosis, by G. B. Webb, M.D.; 1936. Foolscap 8vo, pp. 220. Price: $2.00 net.

CATECHISM SERIES: DISEASES OF THE EAR, NOSE AND THROAT; Second Edition; revised by W. S. Syme, M.B., F.R.F.P.S.; 1936. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 68. Price: 1s. 6d. net.

TREATMENT IN GENERAL PRACTICE: THE MANAGEMENT OF SOME MAJOR MEDICAL DISORDERS; articles republished from The British Medical Journal; 1936. London: H. K. Lewis and Company Limited. Demy 8vo, pp. 258. Price: 8s. 6d. net.

THE SPECIFICITY OF SEROLOGICAL REACTIONS, by K. Landsteiner, M.D.; 1936. Springfield: Charles C. Thomas; London: Baillière, Tindall and Cox. Royal 8vo, pp. 185. Price: $4.00 net.

SYNOPSIS OF THE BRITISH PHARMACOPŒIA, 1932, AND OF THE POISON LAWS, by H. W. Gadd; Thirteenth Edition; 1936. London: Baillière, Tindall and Cox. Medium 32mo, pp. 200. Price: 3s. net.

Diary for the Month.

JUNE 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 24.—Victorian Branch, B.M.A.: Council.
JUNE 25.—South Australian Branch, B.M.A.: Branch.
JUNE 25.—New South Wales Branch, B.M.A.: Branch.
JUNE 26.—Queensland Branch, B.M.A.: Council.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiii, xiv, xv.

FREMANTLE HOSPITAL, FREMANTLE, WESTERN AUSTRALIA: Junior Resident Medical Officer.

PERTH HOSPITAL, PERTH, WESTERN AUSTRALIA: Resident Pathologist and Bio-Chemist.

PUBLIC SERVICE BOARD, SYDNEY, NEW SOUTH WALES: Medical Officer.

ROYAL ALEXANDRA HOSPITAL FOR CHILDREN, SYDNEY, NEW SOUTH WALES: Honorary Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

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